FEB 5 1944

VOLUME 27 · NUMBER 1 · JANUARY 1944

AMERICAN JOURNAL OF OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS

Medical Library

		PAGE
Massive bilateral preretinal hemorrhage	*L. C. Drews and J. Minckler*	1
Epidemic keratoconjunctivitis	*T. D. Allen*	16
Temporal arteritis	*L. T. Post and T. E. Sanders*	19
Military ophthalmology	*W. T. Davis*	26
Emile Javal	*B. Chance*	45
Endophthalmitis phaco-anaphylactica	*R. G. Scobee and H. C. Slaughter*	49
Visual acuity at low brightness-levels	*M. Luckiesh and A. H. Taylor*	53
Orthoptics for the infant squinter	*E. V. Roth*	57
Use of prisms in orthoptics	*C. Burri*	61
Dermoid tumor of the sclera	*L. F. Carter*	67
Technique of tonometry	*M. J. Schoenberg*	70
New suture for muscle-recession operations	*C. B. Foster*	71

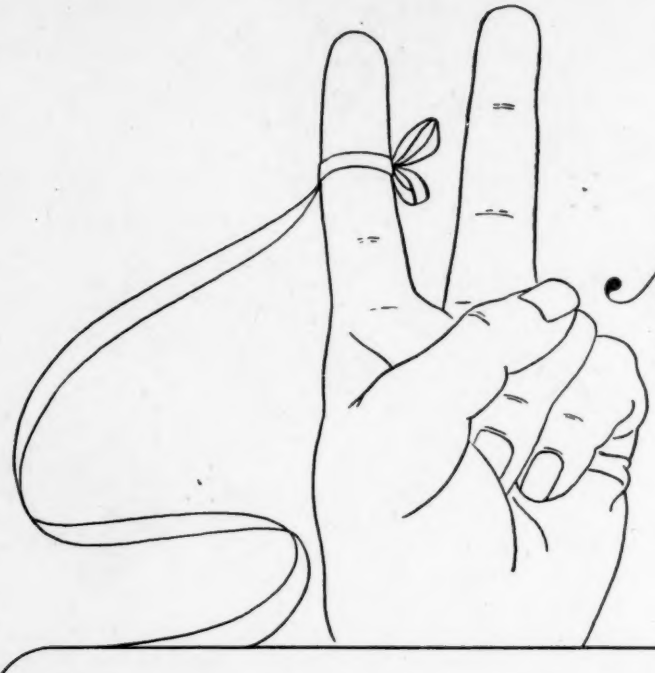
DEPARTMENTS

Society Proceedings	73
Editorials	84
Book Notice	89
Correspondence	90
Abstracts	91
Pan-American Notes	105
News Items	106

For complete table of contents see **advertising** page V

Copyright, 1944, Ophthalmic Publishing Company, 837 Carew Tower, Cincinnati, Ohio

Subscription price in United States ten dollars yearly. In Canada and foreign countries twelve dollars. Published monthly by the George Banta Publishing Company, 450 Ahnaip Street, Menasha, Wisconsin, for the Ophthalmic Publishing Company. Subscription and Editorial Office: 837 Carew Tower, Cincinnati, Ohio. Entered as second class matter at the post office at Menasha, Wisconsin.



Memo for '44

BUY WAR BONDS

Distributors of Bausch & Lomb Ophthalmic Products

Over 36 years experience in serving the profession

Correct interpretation of your prescription

Riggs Craftsmen's expert workmanship

Riggs Optical Company • GENERAL OFFICES: CHICAGO, SAN FRANCISCO

BRANCHES IN PRINCIPAL MID-WEST AND WESTERN CITIES

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 27 JANUARY, 1944 NUMBER 1

MASSIVE BILATERAL PRERETINAL TYPE OF HEMORRHAGE ASSOCIATED WITH SUBARACHNOIDAL HEMORRHAGE OF BRAIN*

WITH CASE REPORT AND PATHOLOGIC FINDINGS

LESLIE C. DREWS, M.D., AND JEFF MINCKLER, PH.D.
Saint Louis

INTRODUCTION

Both subdural and subarachnoidal hemorrhages of the brain may invade the corresponding cavities of the optic nerve and ordinarily will remain localized in that space, if there is no traumatic rupture of the arachnoid that allows the blood to pass into both spaces. Duke-Elder[1] classifies *subarachnoidal hemorrhage* as: (1) traumatic (the occasional case), due to rupture of meningeal blood vessels usually in a basal fracture, and (2) nontraumatic (the usual case), in which the blood always comes from an intracranial origin; either from (a) aneurysm, usually basal, rupturing into the subarachnoid, (b) spontaneous intracerebral hemorrhage bursting into the subarachnoid, or (c) meningeal hemorrhage in blood dyscrasias.

We shall concern ourselves chiefly with hemorrhage into the nerve sheath from a bleeding basal aneurysm. Most of the reported cases are not associated with hypertension or blood-vessel disease; most are precipitated by some temporary increase of blood pressure; most of these aneurysms are in an artery of the first order and most are in the arterior half of the circle of Willis; occasionally the aneurysm may be located in a cortical branch. Usually the aneurysm is congenital and often it is associated with supernumerary blood vessels or other minor congenital anomalies of the circle. Occasionally the aneurysm is thought to be acquired as a result of infected emboli, atheroma, or syphilis (Duke-Elder[2]).

In 1924 Symonds[3] reported a series of 124 cases of "Spontaneous subarachnoid hemorrhage" (that is "spontaneous," or nontraumatic). In only 14 of these 124 cases were the fundi described; 3 of these patients had subhyaloid hemorrhages and 6 had papilledema. In this masterful 27-page article the author divides the cases into four clinical groups, patients in the first of which die suddenly, as in other apoplexies, and those in the last of which (a hypothetical group) have such gradual hemorrhage as to be almost asymptomatic until the grand attack occurs. The other groups are a coma-and- later-meningeal type, and a purely meningeal type.

Premonitory signs and symptoms are often present over many years, and usually are caused by the leakage of varying amounts of blood. Duke-Elder[4] lists them as: (1) Paroxysmal headaches which are severe, sudden in onset, come on after some exertion, and are often associated

* From the Departments of Ophthalmology and Anatomy, Saint Louis University School of Medicine. Read before the Saint Louis Ophthalmic Society, March 27, 1943.

with tinnitus. (2) Coma, then signs of meningeal irritation with delirium, vomiting, restlessness, severe headaches, neck rigidity, positive Kernig, positive Babinski, and paresis of cranial nerves especially the third and sixth. (3) Ocular signs: The extension of hemorrhage into the optic-nerve sheath produces a fairly characteristic picture of papilledema and hemorrhages, an associated palsy of third and sixth nerves in 70 percent of the cases, and moderate proptosis considered due to the hematoma of the nerve. Almost invariably the ocular signs are bilateral although enough unilateral cases are described to emphasize that this need not be so.

Papilledema is the most common finding. Usually it is slight in amount, and only rarely is it entirely absent. It is almost invariably bilateral, and characteristically comes on with great rapidity (Uhthoff, 1901, described it within 30 minutes of onset). The papilledema is of particular interest because it is supposed to be due to the same essential cause as the papilledema of brain tumor. Many of the reported cases show marked hematoma of the nerve sheath, and not a few cases are reported in which definite constriction of the central retinal vein within the distended subarachnoidal space was demonstrated (Dupuy-Dutemps[5]). This obstruction of the central retinal vein within the subarachnoid space as in other papilledemas is supposed to be due to: (1) the increased pressure within the subarachnoidal space, (2) the double angulation of the vein produced by the distention of the subarachnoidal space; (3) the actual axial elongation of the intra-arachnoidal portion of the vein produced by the stretching of the vein between its attachments to pia and arachnoid. In this connection the observation of Dupuy-Dutemps of hemorrhages within the dural sheath due to tearing the elongated vein where it seems rigidly attached to the dura, is of interest.

The retinal hemorrhages. In the great majority of cases the retinal blood vessels are normal. Usually the hemorrhages are small and located near the disc (Duke-Elder[6]), but in four cases proved with serial sections, MacDonald[7] found two with large subhyaloid hemorrhages some distance from the disc. Hale White[8] reported a case with symmetrical, large, brick-red hemorrhages in each eye, about four times the size of the disc and encroaching a little upon it. MacDonald states that *subhyaloid hemorrhages are not necessarily in close contact with the disc,* and that probably the fact that the large retinal arteries usually cross the large retinal veins near the disc may be the factor which in many cases tends to fix the location of the hemorrhages near the disc. According to MacDonald, also, ruptures of the deeper veins would produce punctate hemorrhages, and rupture of the small veins in the fibro-vascular layers would produce striate hemorrhages. All these types of hemorrhages were found in one of MacDonald's cases. MacDonald concludes that *the internal limiting membrane seems to be a definite structure capable of localizing a venous or capillary hemorrhage outside the the vitreous body,* and advances this conception as further proof that these hemorrhages are, in fact, not arterial, since arterial hemorrhage frequently ruptures the internal limiting membrane, allowing the blood to penetrate into vitreous. Occasionally the hemorrhages are subretinal, but some of the old authors called preretinal hemorrhages, "subretinal" (see Hale White's article).

Source of intraocular hemorrhage. No continuity between the retinal and nerve-sheath hemorrhage has been demonstrated in any recent cases. Duke-Elder[9] states that Liebrecht concluded there must be

such continuity, and Hale White had reported the actual continuity. However, Doubler and Marlow[10] also claimed in a case of rupture of an *aneurysm of the internal carotid* with subhyaloid hemorrhage that blood had been forced from the subarachnoid space through nerve substance and lamina cribrosa into the retina, and they had serial sections to substantiate this contention. In such a case the pressure within the subarachnoid might be very high. Paton,[11] who pioneered in investigations of the mechanism of papilledema, said that "subhyaloid hemorrhages in subarachoid brain hemorrhage are the result of direct leakage of blood through the subpial lymph spaces around the lamina cribrosa and through the perivascular lymphatics that surround the central vessels." He believed fully that the subhyaloid hemorrhage directly communicated with the hemorrhage in the subarachnoidal space of the optic nerve. Symonds[12] also concludes that, in his case, blood had been forced through the lamina cribrosa into the subhyaloid space and quotes both Hale White and Doubler and Marlow to show that this may be the source of subhyaloid hemorrhage. In all three of these cases (Doubler and Marlow, Symonds, and White) there was a ruptured aneurysm of the internal carotid artery in which very high pressure might be transmitted to the subarachnoidal space. Because of the question as to the source of hemorrhage, MacDonald investigated his four specimens (in two of which there were large subhyaloid hemorrhages) thoroughly with serial sections and came to the conclusion that blood was not forced from the nerve sheath into the eye. All other reported cases, except those of Doubler and Marlow, Symonds, and White, were conspicuous for the fact that the blood was not forced through the lamina cribrosa.

Riddoch and Goulden[13] state "The ophthalmoscopic appearances conform very much with our ideas of venous engorgement of the retina which are seen in its *most marked instance in cases of thrombosis of the central retinal vessel.* The obstruction in hemorrhage of the optic nerve sheath is sudden, thus causing appearances similar to those seen in thrombosis of the central retinal vessel although the *results are not so serious as the obstruction is neither so complete nor so prolonged as in thrombosis.*"

Riddoch and Goulden[14] also state that post-mortem evidence indicates that whether or not there will be ocular hemorrhage in cases of subarachnoid hemorrhage depends upon (1) the severity of the hemorrhage, (2) situation of the aneurysm, and (3) presence of scar tissue from previous hemorrhages (arachnoidal adhesions in the cisternae basalis and chiasmatis). Thus if scar tissue prevents the hemorrhage from passing down the optic-nerve sheath, there may be no intraocular signs. If only one optic-nerve sheath is "patent" the intraocular signs, if any, will be present only in that eye.

Diagnosis of hemorrhage into the subarachoidal space of the optic nerve may be made with considerable certainty when there are papilledema, retinal hemorrhages, palsy of third and sixth nerves, and a history of either head trauma or of a sudden seizure with signs of increased intracranial pressure, or with signs of meningeal irritation. The diagnosis is confirmed by lumbar puncture and the finding of bloody spinal fluid (Duke-Elder[15]).

Case report

Mrs. D. B., white, aged 36 years, a patient of Drs. C. J. Vollmar and R. A. Kinsella, entered St. Mary's Hospital on September 6, 1943, at 10:00 a.m. and died September 30, 1943. At 9:00 a.m. while

preparing for church the patient suddenly complained of severe frontal headaches, and had to be led to a sofa, whereupon within less than five minutes she lapsed into a coma with convulsions in which there were periods of rigidity of extremities, jaws, and back. Her face became fiery red and she vomited profusely. She never regained consciousness. Dr. Vollmar was called and sent the patient to the Hospital within the hour.

Past history. When the patient was 12 years old she and her sister had St. Vitus's dance, from which they both recovered completely within several months. At the age of 14 years, she wandered about for two days without knowing who she was, then suddenly recovered. She had had frequent severe headaches since childhood. Seven years ago the patient had what was diagnosed as meningitis, with stiff neck and vomiting. No lumbar puncture was done. Since that time she had had many more headaches than before. The headaches were very severe frontal headaches associated with increasingly severe nausea and vomiting. She frequently was awakened with severe headaches. She was diagnosed hypertensive 2½ years ago, and one year ago was told not to become pregnant again because of hypertension. She had a fall from a horse 20 years ago without known injury. No other injuries were known to her husband. She had had no operations. There was no definite ocular history, and no history of diplopia.

Family history. Grandfather died of paralysis. Mother died of diabetes. Father and brother both died of coronary disease. One uncle has had a bad heart since youth.

Marital history. One child, nine years of age. Otherwise negative.

Physical examination was made one hour after onset of the present illness. The patient was a well-developed and -nourished woman of 36 years, comatose, face markedly flushed, breathing rapid and shallow. There was moderate tonic convulsive rigidity. Her jaws were clamped tightly on a gag. The eyes were closed, the pupils moderately dilated and fixed to light and accommodation. Ophthalmoscopic examination by the interne was recorded: "The right fundus shows a choked disc. Left fundus cannot be seen." The blood pressure was 200/104, pulse 98 and regular, respiration 40. Examination of chest showed generalized loud moist rales, which obscured the heart sounds. Babinsky reflex was positive on both sides.

PROGRESS NOTES

September 7th. Patient had purposeful movements of the limbs and occasionally tried to talk. Right Babinsky had decreased and left was negative.

September 8th. The patient's blood pressure was running 100-112/60-80, in both arms. She had severe pain in the right side of the neck posteriorly and winced when this area was touched.

September 9th. She had been moving hands a great deal scratching furiously and beating both hands against addomen. Her right arm was more active than her left. Blood pressure was now 202/116. Pulse and temperature were normal.

September 11th. Ophthalmoscopic examination recorded by interne: "Numerous hemorrhages are present over the right retinal surface. Left eyeground not visualized." Temperature 102.6°F. respiration 22, pulse 90, blood pressure 182/100.

Spinal tap showed pressure 380 mm. of water. When 7 c.c. of bloody spinal fluid was removed the pressure dropped to 135 mm. of water. General condition remained about the same.

Examination by one of us (L. C. D.): "The pupils react well to light. The

eyes are turned up and out. Homatropine 1 percent produced dilatation to 8 mm.

"Ophthalmoscopically the entire posterior pole of each eye for a width of about 10 D.D. is covered by a large retinal or preretinal hemorrhage of uniform dark brick-red color through which no fundus details are visible. The hemorrhage seems centered over the area where the disc should be. There is a sudden sharp transition from hemorrhage to the normal retina in the periphery, the edge of the hemorrhage is rather smooth, and the outline is roughly circular as in preretinal hemorrhage. The media are clear. The blood vessels in the visible normal retina are free of arteriosclerosis; the veins are markedly tortuous but only moderately engorged."

Diagnosis: "Massive subarachnoidal hemorrhage of the brain extending down along the optic nerve. It is believed that usually such hemorrhage of the eye is due to compression of the central retinal vein within the subarachnoidal space of the nerve. Very frequently the cause is congenital aneurysm of the basal arteries. Occasionally such aneurysms have been attributed to meningitis of which there is a possible history here."

September 16th. Lumbar puncture: spinal fluid bloody, pressure 85 mm. of water. One cubic centimeter of fluid withdrawn.

September 17th. Ophthalmoscopic examination (L. C. D.), with dilatation ad maximum. "All the gross hemorrhage in each eye has been absorbed. Cannot make out even the location of the discs through a dense grayish haze which seems centered over the disc area to a width of 3 to 4 D.D. The obscured area is much smaller than the hemorrhage first seen on September 11th. The peripheral retina uncovered by the absorbed hemorrhage seems normal."

September 22d. Ophthalmoscopic examination (L. C. D.) showed no change over September 17th. Lumbar puncture showed the intraspinal fluid pressure to be 235 mm. water. When 10 c.c. of bloody spinal fluid were withdrawn, the pressure dropped to 180; then 8 more c.c. were removed, after which pressure dropped to 85.

September 29th. Ophthalmoscopic examination (L. C. D.). "No change from September 17th and 22d." Twelve hours later the patient had 6 to 8 convulsions; blood pressure was 240-290/110-160. Pupils were dilated but reacted to light, Babinsky reflexes negative. During intervals between convulsions the patient relaxed completely, pupils constricted and were fixed to light. Convulsions continued and *two hours later,* the patient regurgitated a mouthful of fluid, then immediately aspirated it, became cyanotic and respiration became labored and very irregular. Trendelenburg position, oxygen, artificial respiration, coramine, all were ineffective. Respiration gradually ceased, cyanosis increased, and within a minute after respiration ceased, the heart stopped.

POST MORTEM. September 30th, 8:30 a.m.

GENERAL. Rigor mortis four plus. Hypostasis slight. Body is well developed and well nourished. The heart weighs 360 grams, shows no evidence of infarction or scarring. The valve cusps are delicate and competent. The coronary arteries are straight and patent and show minimal atheromatous change. There is grade I (minimal) atherosclerosis of the aortic lining. Microscopically, there is hypertrophy of myocardial fibers, and there are a few lymphocytes in scattered subendocardial foci. The blood-vessel walls are not thickened and there is no evidence of inflammatory reaction. Hyaline change in the walls of the afferent glomerular arteries of the kidney is evident and there is minimal medial hypertrophy of small

arteries. There is marked congestion of the pulmonary vasculature with apparent thickening of interstitial lung tissue. The remaining general findings are minor and irrelevant. Death is considered to be respiratory, incident to the cerebral pathology.

Cerebrospinal system: The brain with membranes exclusive of dura weighs 1,370 grams. The gyri are somewhat flattened but there are no basal-pressure markings (figs. 1 and 2). The arachnoidal membrane is thickened diffusely and has a bluish cast. There is no fresh or old subarachnoid blood in evidence over the vertex or sides. The superficial cerebral veins are moderately distended. There is clotted hemorrhage in a subarachnoid position over the medial and inferior aspects of the left frontal lobe. The basal arachnoid is conspicuously thickened and dissects away with great difficulty. The cisterna cerebellomedullaris is choked with hemorrhage but shows no increase in trabecular consistency. Frank blood is in evidence in a subarachnoid position over the foramina of Luschka. The cisterna superius and the cisterna venae magnae cerebri are similarly engorged. The cisterna pontis shows patches of old hemorrhage with marked tenacity of the subarachnoid trabecula. The increase in trabecular consistency in the entire basal cistern (c. interpeduncularis, basalis proper, chiasmatis, and fissurae lateralis cerebri) is marked. This space (fig. 2) dissects with unusual difficulty and incorporates old blood diffusely throughout the trabecular reticulum. This network is interpreted as being not wholly related to the terminal hemorrhage but *incident to previous basal pathology* (arachnitis from

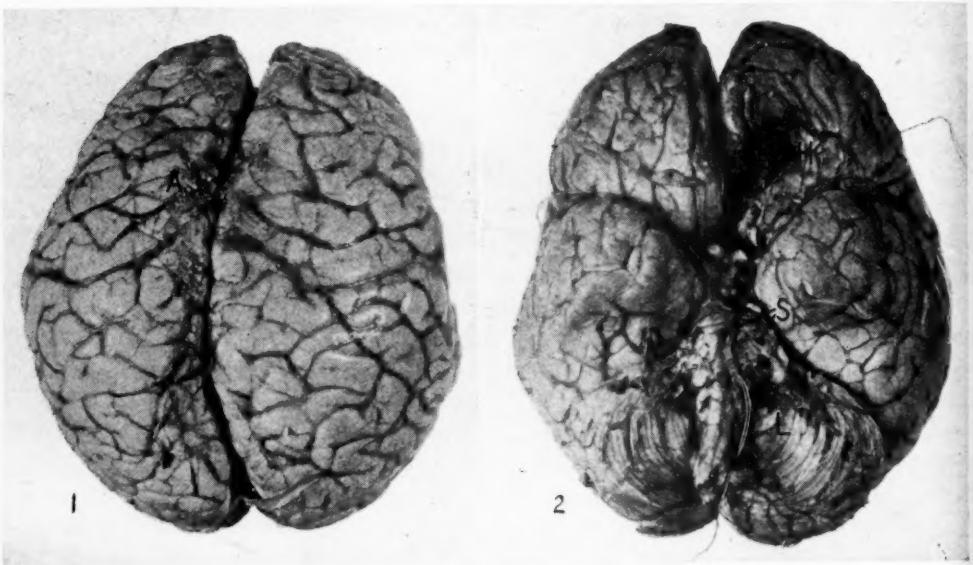


Fig. 1. (Drews and Minckler). Superior view of brain. Note flattening of gyri, absence of free blood in the obliterated subarachnoid space. A, arachnoid granulation.

Fig. 2. Inferior view of brain. Note the large hemorrhage in left frontal pole (H), scattered subarachnoidal blood over the base and stem (S), thickened arachnoid and hemorrhage at the foramina of Luschka (L).

recurring subarachnoid hemorrhage or possibly meningitis—see history).

On the inferior surface of the left frontal lobe there is a gross hemorrhage measuring 2.5 cm. in diameter which dissects into the left cerebrum (fig. 2). Dissection of the basal region reveals the source of the hemorrhage in the circulus arteriosus. There is a gross defect of the anterior communicating artery (fig. 3). The right anterior cerebral artery continues past the anterior communicating branch in a normal manner. One small lateral branch is given off at the level of the communicating artery and the distal ramifications are in normal order. The left anterior cerebral artery has a stellate distribution at this level. Laterally, four small vessels are contributed to the brain substance. Distal to the communicating branch the left artery divides immediately into superior and inferior branches. The anterior communicating artery is disposed in two closely related divisions, an anterior and a posterior bridge. The latter arises as a small branch of the right, and a dilated conical segment of the left anterior cerebral arteries. The anterior bridge has a wide base on both longitudinal vessels and fuses widely in its central part with the posterior bridge. The middle fused part extends in an infundibuliform manner superiorly and to the left, terminating in a rounded bulb with a firm white consistence suggestive of a sclerotic plaque. Inferiorly, the stalk of the aneurysm is in evidence with an aperture communicating with the interior of the artery and

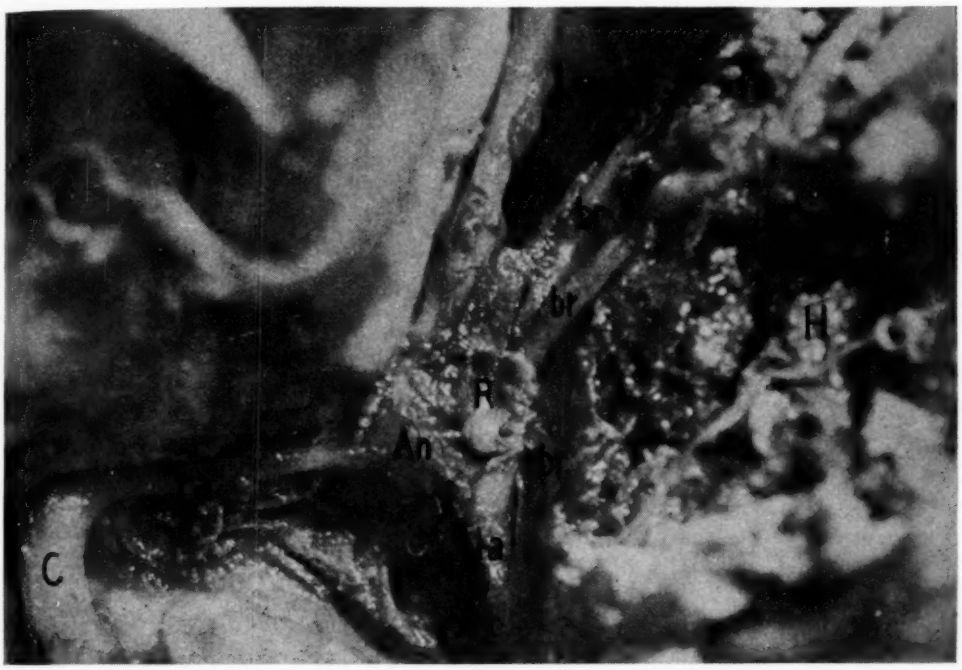


Fig. 3 (Drews and Minckler). Enlargement showing right carotid artery (C), right and left anterior cerebral arteries (ra, la), the aneurysm at the site of the anterior communicating artery (An), the point of rupture of the aneurysm (R), the stellate branching of the left anterior cerebral (br), and the invading frontal hemorrhage (H).

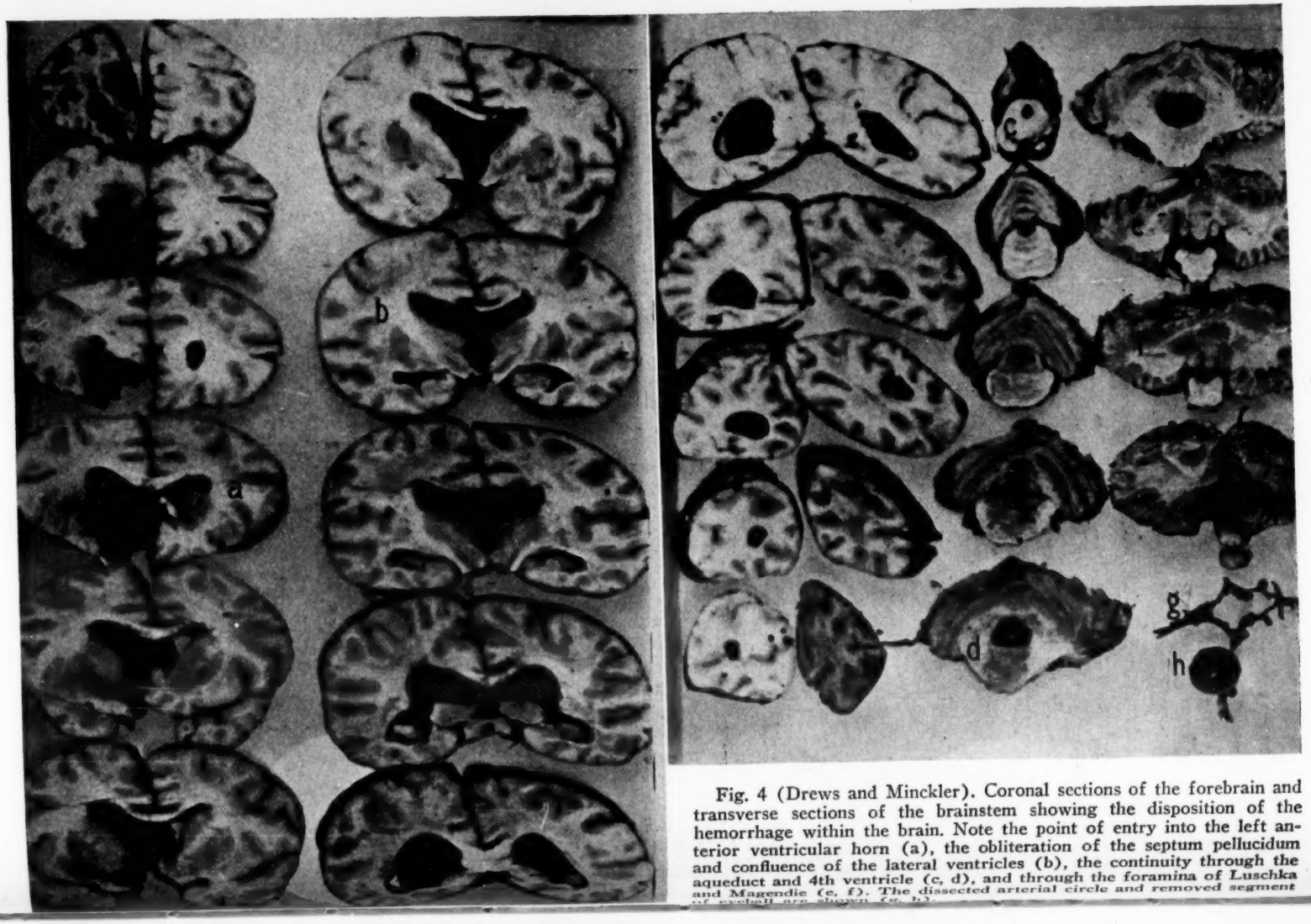


Fig. 4 (Drews and Minckler). Coronal sections of the forebrain and transverse sections of the brainstem showing the disposition of the hemorrhage within the brain. Note the point of entry into the left anterior ventricular horn (a), the obliteration of the septum pellucidum and confluence of the lateral ventricles (b), the continuity through the aqueduct and 4th ventricle (c, d), and through the foramina of Luschka and Magendie (e, f). The dissected arterial circle and removed segment of cerebellar artery shown (g, h).

pointing anteriorly and to the left (fig. 3). It is from this aperture that the hemorrhage had its origin, dissecting superiorly into the substance of the left frontal lobe.

Gross coronal sections (fig. 4) reveal the course of the hemorrhage throughout the ventricular system. Entering the left anterior ventricular horn the blood perfused the entire system, establishing confluence of the lateral ventricles by destruction of the septum pellucidum, and issuing through the lateral and median apertures of the fourth ventricle to appear in the subarachnoid space of the brain stem and cord. Undoubtedly, the arachnitis in the basal cistern from previous seepage of the aneurysm influenced the course of the hemorrhage through brain substance and the intensity of the packing of blood in the ventricular system.

MICROSCOPIC. Sections through the aneurysm show a uniform defective wall with great hypertrophy and absence of an elastic membrane (fig. 5, cf. Forbus[16]).

Meninges and brain: The arachnoidal membrane is thickened, cellular, and compressed over levels including vertex, lateral surface, base, stem, and cord. Free blood is present except as noted grossly. Meningothelial phagocytic activity is pronounced in the subarachnoidal space of the spinal cord, less marked at the levels

Fig. 5 (Drews and Minckler). Low-power view of section through floor of ruptured aneurysm (An), the superior infundibular extension of the aneurysm (San), the left anterior cerebral artery (la). Note the defective wall of the aneurysm (Aw).

of the stem and base. At these levels the trabecular network is dense, with free red blood cells and lymphocytes in the meshwork and relatively few macrophages with pigmented débris. The pia-glial membrane is thick and sclerotic throughout. The intramedullary pathology is not remarkable. There is no evidence of glial, free-cell, or vascular activity suggesting an inflammatory process. Cell changes are nonspecific. The intramedullary small arteries show a minimal medial hypertrophy. Edema is evident but not marked in cerebrum and stem, is absent in the spinal cord and minimal in the optic chiasma. The edema does not extend into the optic nerves. Transverse sections through the latter to within 1 cm. of the bulb reveal *free blood in the subarachnoid space in diminishing quantity as the inspection is carried distally.* Close to the chiasm there is free blood with numerous macrophages. Red cells are present in progressively smaller amounts to about the midpoint of the stalk bilaterally (fig. 6). Distal to this point no free cells have been found but *old hemorrhage is indicated by the presence of scattered macrophages containing pigmented débris.* The arachnoidal space close to the bulb is dilated and shows increased density of trabecula (fig. 7).

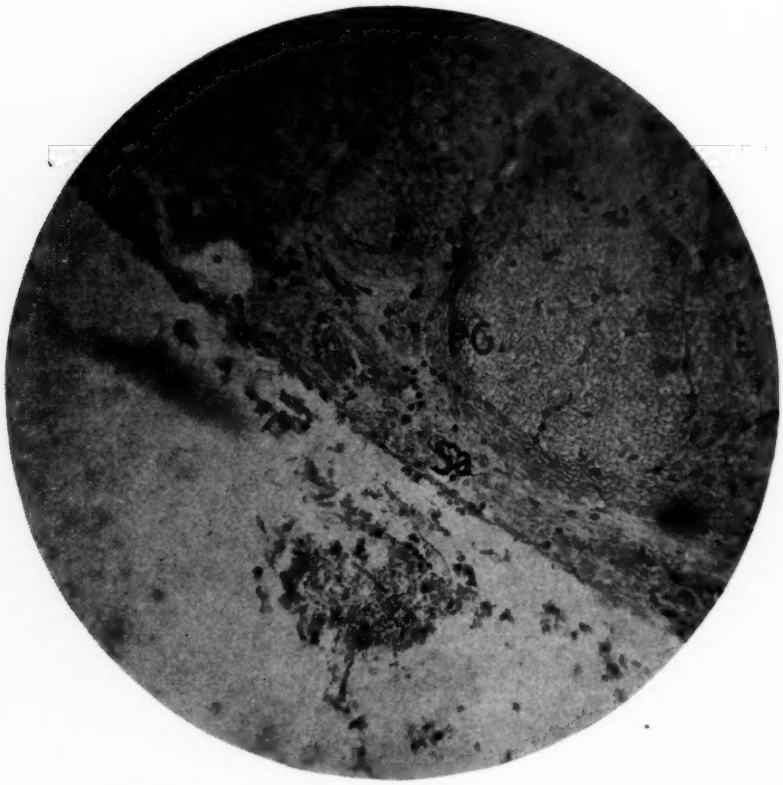


Fig. 6 (Drews and Minckler). Section through the left optic nerve 2 cm. from the chiasm showing free blood in subarachnoid space (Sa). PG, pia glial membrane.

Longitudinal sections through one papilla show dilated central vein and moderate lateral shift of the retina. There is a slight forward bulge of the anterior com-

ponent of the lamina cribrosa. Distortion of fibers entering the nerve is not marked (fig. 7). There is an extensive intraretinal hemorrhage (fig. 8). This has dissected into the anterior part of the nerve fiber layer and at all points appears to be separated from the vitreous by a thin, but distinct membrane of varying cellularity (fig. 8). Careful search reveals no actual breach in the wall of the retinal vein but the wall is obviously defective adjacent to the hemorrhage with evident diapedesis. The nerve-fiber layer is edematous and the ganglion cells show a vacuolar change with eccentricity and pyknosis of nuclei.

Fig. 7. (Drews and Minckler). Low-power view of one half the papilla showing lateral shift of retina (R), moderate distortion of nerve fibers entering the optic nerve (N), dilated central retinal vein (V), dilated subarachnoid space (Sa). The anterior fibers of the lamina cribosa bulge forward slightly. Note absence of blood in an interfasicular position at the level of the lamina cribosa. The relative position of intraretinal hemorrhage (H) is shown.

Comments

When I first saw this case I was struck by the fact that the retinal hemorrhages were far more extensive than any I had ever seen in cases of thrombosis of the central retinal vein, or, for that matter, anywhere else. One might think, as actually stated by Riddoch and Goulden and quoted before, that where the vein is completely blocked, as in thrombosis, we should see the most extensive venous or capillary hemorrhage possible. Komoto[17] described a preretinal hemorrhage 7 by 10 mm. in size (that is about 5 by 7 D.D.) centered over the macula, and not in contact with the disc, in a fatal case of pur-

pura. I have not been able to find any case in the literature where the retinal hemorrhage was as extensive as in the case here described. Of course, I suppose where hemorrhage is as extensive as in this case it is unusual to find it limited to the retina; in most cases, undoubtedly, it bursts into the vitreous, making ophthalmoscopic examination impossible.

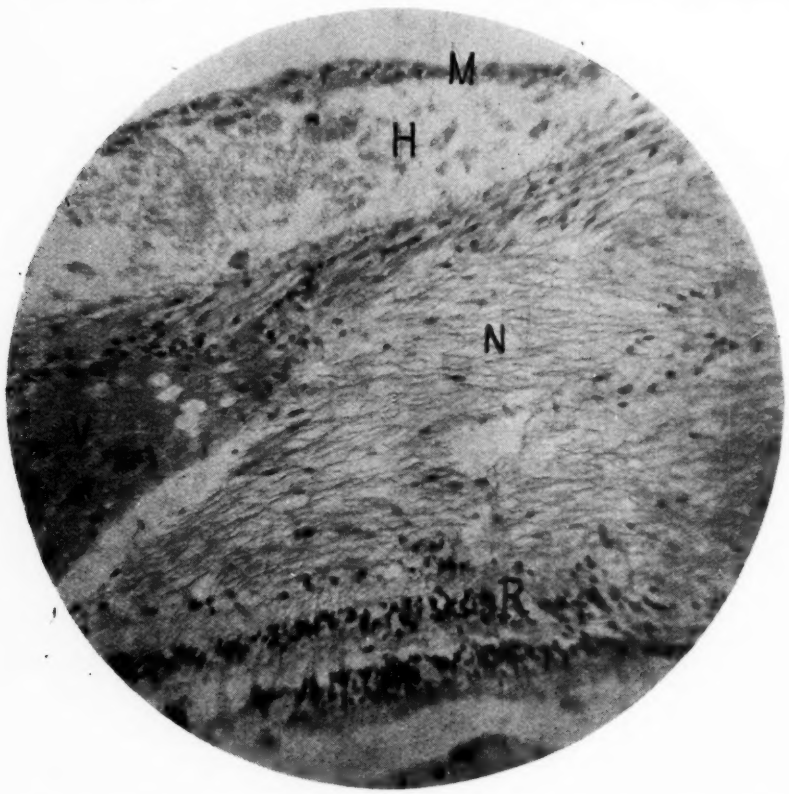


Fig. 8 (Drews and Minckler). High-power view showing retina (R), edematous nerve-fiber layer (N), intraretinal hemorrhage (H), separated from the vitreous by a cellular membrane (M). A retinal vein with a defective wall adjacent to the hemorrhage is shown (V).

It seemed to me that if blood ever were forced through the lamina cribrosa into the eye, this would be a case where such a finding could be expected. In this connection one might wonder why a pressure sufficient to force blood into the eye through the nerve substance and lamina cribrosa would not immediately rupture the hyaloid, forcing its way into the vitreous, even if the blood does travel over the perivascular spaces. If such a hemorrhage, coming through the disc, is to be localized as a preretinal hemorrhage it must be that it is easier to extravasate beneath the internal limiting membrane than to break it and the hyaloid. Moreover, if the blood came through the disc one might expect to find some blood in the canal of Cloquet.

The hemorrhages were described in the first examination as "retinal or preretinal." It was thought that even if they were preretinal the gravitational effect diagnostic of preretinal hemorrhages would be absent because of the patient's

prone position. The smooth outline between hemorrhage and normal retina also suggested preretinal hemorrhage. There is considerable confusion between the terms preretinal and subhyaloid. I have used them interchangeably. In this case, which had some of the characteristics of preretinal or subhyaloid hemorrhage, the hemorrhage actually seems to be entirely *beneath the internal limiting membrane,* which is intact but separated from the nerve-fiber layer by some old hemorrhage and detritus. Some authors have reported that preretinal hemorrhages actually lie beneath the internal limiting membrane; others have found the preretinal hemorrhage located between the internal limiting membrane and the condensation membrane of the vitreous. Duke-Elder[18] says that "usually the blood is poured out beneath the internal limiting membrane which it finds little difficulty in stripping off and raising up so that it forms a film immediately *overlying the retina;* sometimes, however, the internal limiting membrane may be ruptured in which case the hemorrhage forms a similar film between it and the vitreous; and occasionally blood bursts through the face of the vitreous and forms a diffuse opacification in the gel." If the internal limiting membrane is actually formed by the expanded ends of the sustentacular cells of Müller it seems strange that it could easily be detached. Unfortunately only the posterior half of the one eye was removed; therefore, the eye specimen is not all that could be desired. Both optic nerves and the chiasma, however, were removed.

From a clinical standpoint it seems certain that since all the gross *retinal* hemorrhage which had been so extensive was resorbed in only six days or less, the blood was situated in a location having very good circulation. It has often been remarked that a large preretinal hemorrhage may be absorbed (and unfortunately recur) with surprising rapidity, but if the hemorrhage actually were situated between the internal limiting membrane and the vitreous, one would not think it likely that it could be rapidly absorbed. Unless there were multiple large tears through the internal limiting membrane it is difficult to believe that there could be a rapid absorption of a truly preretinal hemorrhage. However that may be, it seems certain that, theoretically at least, a large hemorrhage entirely beneath the internal limiting membrane could be rapidly absorbed, whereas a similar hemorrhage between the internal limiting membrane and the vitreous probably could not. In the possible rapidity of absorption, therefore, we may have a suggestion as to the exact situation of a hemorrhage, which appears to be "preretinal."

It seems unfortunate to call hemorrhages beneath the internal limiting membrane either "preretinal" or "subhyaloid" since the hemorrhage really is intraretinal. An intraretinal hemorrhage should be expected to give rise to more retinal damage than a truly subhyaloid one, therefore there is clinical value to differentiation between the two types of "preretinal" hemorrhages. However there seems to be no way in which the two types of hemorrhages can be distinguished ophthalmoscopically, so the use of one term for both conditions seems advisable.

At the autopsy I was rather chagrined to find no gross hematoma of the optic-nerve sheath. Microscopically, we found no hemorrhage in the sheath; there was a small amount of blood in the sheath near the chiasm. This raised the question whether or not there ever was any subarachnoid hematoma of the nerve. All we can say is that we did find moderate hydrops and considerable detritus in the

subarachnoidal space of the nerve, *which was due to hemorrhage* (see pathology report). When one recalls that the very large preretinal hemorrhage was all absorbed, so far as could be observed with the ophthalmoscope, within 6 days, it seems quite certain that even an extensive subarachnoidal hemorrhage into the nerve sheath could have been absorbed within 24 days, the period of time between the onset and the fatal attack.

If the first subarachnoidal brain hemorrhage produced a hematoma of the nerve, why did the fatal attack 24 days later fail to do so? There may have been minor attacks in between the first and fatal attacks as well, as indicated in the history. Perhaps the fatal hemorrhage did not have time to penetrate into the optic-nerve sheath; but probably what happened was that the extensive adhesions in the cisterna basalis and chiasmatis caused by the first hemorrhage prevented the fatal hemorrhage (and any others which may have intervened) from reaching the optic-nerve sheath.

In some personal discussions, there has been disagreement that a large preretinal hemorrhage in each eye associated with subarachnoidal hemorrhage of the brain justified the diagnosis of subarachnoidal hemorrhage of the optic nerve. However, I believe that the literature substantiates the validity of my conclusion that large preretinal hemorrhages in each eye in a patient not the victim of general vascular disease, associated with the typical spinal-fluid findings in subarachnoidal hemorrhage, do justify the diagnosis of subarachnoidal optic-nerve hematoma, even though in this case the actual hematoma was not found to be present 24 days after the onset. None of the reported cases in the literature failed to show such a hematoma; but also none of the reported cases had the time intervals reported here.

The specimen shows slight but definite papilledema; whether this was present beneath the "preretinal" hemorrhage when first noted no one could possibly say. I have no clinical doubt that it was. No one could possibly deny, however, that the papilledema could have been produced by the supposed later hemorrhage with increased intracranial pressure, or even by the fatal hemorrhage.

The autopsy revealed only minimal arteriosclerosis, yet the patient had been diagnosed hypertensive by two competent practitioners. It is an interesting question to ask whether she had periods of acute hypertension related to minor attacks of bleeding from the aneurysm, similar to the acute hypertensive attacks she had while in the Hospital. The sclerosis of the renal arterioles seemed sufficient to account for a hypertension.

The crux of the problem of massive prerctinal hemorrhage associated with hematoma of the optic-nerve sheath in subarachnoid hemorrhage is that such massive hemorrhage is so unusual in papilledema associated with brain tumor. What is there about subarachnoidal hemorrhage which favors preretinal hemorrhage? Certainly we would not think that the pressure in the subarachnoidal space would be so very different in the two conditions, and even if it were very different it would still not account for hemorrhages more extensive than those usually seen in actual thrombosis. Of course, in speed of development the two conditions may differ. The more one ponders this the more sympathy one has for Liebrecht, Doubler and Marlow, Symonds, White, and Paton's opinions, which apparently we must discard, that the retinal hemorrhage comes directly from the nerve-sheath hemorrhage. The fact that the pre-retinal hemorrhage may not be in contact with the disc at any time of its development as well as the absence of his-

tologic proof, makes this opinion seem untenable.

Conclusions

1. A case of very extensive bilateral preretinal hemorrhage, associated with subarachnoidal hemorrhage from a congenital aneurysm of the communicating branch of the anterior cerebral artery, is reported. When examined pathologically, 24 days after the onset of the brain hemorrhage, only the remains of hematoma were found in the subarachnoidal space of the optic nerves. It is concluded that hematoma had been present but was absorbed.

2. It is suggested that if a "preretinal" hemorrhage absorbs very rapidly the hemorrhage probably is situated beneath the internal limiting membrane where the circulation may be excellent. Such a hemorrhage really should be considered intraretinal, since the internal limiting membrane is considered a part of the retina.

3. After one or more subarachnoidal hemorrhages, adhesions may form in the cisterna basalis on one or both sides; a later hemorrhage may then fail to produce ophthalmoscopic signs in one or both eyes.

4. As far as I can find this case presents the largest preretinal type of hemorrhage described in the literature (about 10 disc diameters, each eye). There was no evidence that blood had been forced from the supposed hematoma of the nerve sheath through the lamina cribrosa.

5. Why do massive preretinal hemorrhages occur in subarachnoidal hematoma of the optic-nerve sheath frequently, and rarely in ordinary papilledema?

References

[1] Duke-Elder. Textbook of ophthalmology. Saint Louis, C. V. Mosby Co., 1941, v. 3, p. 2939.
[2] ———. Ibid., p. 2940.
[3] Symonds. Quart. Jour. Med., 1924, v. 18, pp. 93-107.
[4] Duke-Elder. See reference 1, p. 2941.
[5] Dupuy-Dutemps. Ann. du Ocul., 1914, Mar.
[6] Duke-Elder. See reference 1, p. 2943.
[7] MacDonald. Trans. Amer. Ophth. Soc., 1931, v. 29, p. 418.
[8] White, Hale. Trans. Clin. Soc. London, 1895, v. 28, p. 5.
[9] Duke-Elder. See reference 1, p. 2943.
[10] Doubler and Marlow. Arch. of Ophth. 1917, v. 46, p. 533.
[11] Paton, Leslie. Proc. Roy. Soc. Med., v. 18, pt. 2.
[12] Symonds. Quart. Jour. Med., 1924, v. 18, p. 107.
[13] Riddoch and Goulden. Brit. Jour. Ophth., 1925, v. 9, p. 224.
[14] ———. Ibid., p. 225.
[15] Duke-Elder. See reference 1, p. 2943.
[16] Forbus, W. D. Bull. Johns Hopkins Hosp., 1930, v. 47, pp. 239-284.
[17] Komoto. Klin. M. f. Augenh., 1912, v. 50, p. 309.
[18] Duke-Elder See reference 1, p. 2600.

EPIDEMIC KERATOCONJUNCTIVITIS FROM A SUBJECTIVE VIEWPOINT*

THOMAS D. ALLEN, M.D.
Chicago

The present epidemic of keratoconjunctivitis, although slightly different from certain epidemics described under the heading "Superficial punctate keratitis," should, in my opinion, be considered in the same category. The Editor of the American Journal of Ophthalmology (1943, v. 26, Feb., p. 199) agrees with this opinion. I have read many of the articles on both subjects. In several of them reference is made to the fact that clinical assistants became infected, but nowhere could I find that a physician had written of the disorder from the subjective standpoint. For this reason it has seemed important to bring before you a slightly different angle of the problem than the purely objective one, which has been adequately presented in numerous recent papers. Also, it has seemed to me expedient to personalize the danger to physicians, and especially ophthalmologists, of infecting their own eyes. One of our Chicago ophthalmologists fell prey to the disease last summer, but very few others knew about it until after I contracted it early in January. Since my illness I have tried to spread the gospel of meticulous cleanliness from Chicago to Mexico City.

The objective signs need not be reviewed here in detail; briefly they are a gradually increasing conjunctival hyperemia and folliculosis, moderate to severe conjunctival edema, some sticky secretion, no pus, occasional pseudo-membrane, slight photophobia, considerable edema of the lids, and regional adenopathy; later often superficial corneal spots varying greatly in size, density, and numbers; seldom iritis. There is a gradual return to normal, as a rule in three to six weeks, except that the corneal spots linger and usually interfere with visual acuity for several months.

Subjectively, I have had a peculiar mixture of the scientific attitude of the practicing physician with the irritation of the restless patient, which is certainly not scientific. I found myself making my own diagnosis (a most dangerous procedure) and wanting all the corroboration I could get. I did what physicians universally deplore and discountenance; that is, I consulted many physicians and compared their diagnoses and recommendations.

I had seen, December 24 and 28, 1942, a patient (E. P. E.—D1369) with a most peculiar conjunctivitis. He had been in the hospital under the care of a very competent ophthalmologist for about a week, but his doctor had been called out of town and he came to me. In addition to the conjunctivitis, which was moderately severe but without much edema, he had a definite aqueous beam, seen with the slitlamp, and some fine precipitates on Descemet's membrane. I looked carefully at the cornea with the slitlamp, and I believe I would have seen any superficial corneal spots if they had been present. On January 12, 1943, after he had seen several other ophthalmologists, he went to Dr. H. Gradle's office, and by that time he had developed the corneal opacities characteristic of the disorder.

About January 10th (Sunday)—that is, 13 days after I last saw E. P. E.—I noticed a slight watering of my own right eye, and the following day a stickiness of the eyelids. I have such sensations one or twice a year, and I have found

* Presented before the American Ophthalmological Society, at Hot Springs, Virginia, June, 1943.

a single application of 1-percent silver nitrate usually suffices to restore the tissues to normal in 24 hours. This time, however, the situation did not improve but was even worse on January 12th (Tuesday) at which time, by coincidence, I attended a luncheon at which the subject of this epidemic was discussed. That evening I went to a nearby ophthalmologist, who made a smear and a culture; the clinical appearance was that of an ordinary conjunctivitis but without much discharge; the laboratory report was "no organisms found."

The next day, January 13th, the eye was distinctly worse. I called Dr. Peter Kronfeld, who admitted it might be the epidemic form, and we decided I should go to the hospital to minimize the danger of contagion at home, and so that studies could be more conveniently conducted. The laboratory report of scrapings of the conjunctiva was: "few staphylococci; few larger structures which look like staphylococcal cells; a number of eosinophiles." On the following day a more critical study was made of another smear; several observers noted "a preponderance of monocytes." About January 14th (Thursday) the eyelids began to be heavy, especially the upper one, and the following day subconjunctival hyperemia was marked and the right preauricular gland was palpable.

One-percent, later 0.25-percent, formaldehyde ointment was used in the left eye to prevent, if possible, its involvement; and in the right eye to see if it would have any effect. It was not, however, used consistently because of the discomfort it caused. I dropped 5-percent sulfathiazol solution into each eye six or seven times a day for the first week; also aqueous mercurochrome 1-percent several times a day for three days, 13th, 14th, 15th. Warm boric-acid compresses were used at first, but cold compresses seemed much more comfortable. Foreign-protein therapy was received as follows: (a) Proteolac 5 c.c. intramuscularly, January 14th and 15th; (b) 50,000,000 triple typhoid vaccine intravenously, January 16th—it produced a chill and fever (temp. 101.6°F.) and what the colored folk call "a misery" for 36 hours.

I made it a point to have a paper handkerchief for the right eye on the right side of the bed or chair, and one for the left eye on the left side; two towels also and two handkerchiefs; but I caught myself wiping the left eye with the same towel and handkerchief I had used for the right eye, and this not just once but several times! It was most difficult to develop suddenly an entirely new technique for cleaning my face!

About January 18th the left eye began to tear and the next day a slight sticky secretion was present. Within a day or two the left preauricular gland became tender. It was hard to say just when it became tender; I felt for it many times every day; the onset of tenderness was almost imperceptible.

On the 23d of January minute subepithelial corneal spots were found in the right eye by Drs. Harry Gradle, Sanford Gifford, and others. Up to this time the visual acuity had not been affected. On January 18th Dr. Kronfeld had sent a request to Dr. Sanders at the Presbyterian Hospital, New York City, for some convalescent serum. This was not immediately available; it seemed much longer than the four days it was before it arrived on January 22d. On that day I was given 50 c.c. intravenously; the dose was repeated the next day, and that evening I started to Texas, as it was quite evident I would be out of circulation, as far as my practice was concerned, for another month. On the train I found it difficult to read, and my morale was pretty low when, between trains at Saint Louis, I called on Dr. Hayward Post. The following day newsprint and the menu could be read only with the help of a

+13.00D. magnifier. Tobacco smoke was unbearable, so I had to stop smoking and keep away from others who smoked.

Two days later, January 27th, I arrived in Houston and immediately saw Dr. Everett Goar. He found my visual acuity varied between 18/200 and 20/100; it was not constant and it was about equal in the two eyes. There were still some preauricular tenderness and moderately severe hyperemia of the eyes, but the edema was less. Dr. Goar started me on vitamin C intravenously (cenolate, an Abbott preparation of ascorbic acid), 200 mg. daily. At that time it was very difficult to read, even with a magnifier, and I had to approach to within 10 to 12 feet of a street sign to read it. Even the spots on playing cards were not distinguishable beyond 12 inches. All lights were multitudinous and extraordinarily dazzling and confusing. Neither a single nor a multiple pinhole disc was of any avail in reducing the discomfort from the lights, although dark glasses were helpful. But even with this poor vision I had no difficulty in crossing streets or on uneven pavements.

After the first two treatments it seemed my visual acuity improved, and I thought I could notice the difference within an hour after a treatment. After the fifth treatment the vision was about 20/70, occasionally even better, and I began to wonder if I could get some pleasure out of the experience—possibly a trip to Mexico City even!—that is, my morale rose. But from the time I left Houston the progress was so slow and so often there would seem to be a regression (fresh spots?) that I became very much discouraged and arranged for immediate return to the States in spite of the fact that Dr. Silva reassured me that all would soon be well. Dr. Ramon Castroviejo saw me just as I was leaving Mexico City and did his best also to cheer me up.

Even back in Houston, February 6th to 12th, the improvement was slow. Acuity varied between 20/40 and 20/30+. It seemed to vary from hour to hour. I attended a clinic with Dr. Edward Griffey and had to admit I did not see details clearly enough to hazard a diagnosis. However, in the dark room I found, with the electric ophthalmoscope, that my acuity was almost normal. Again my morale picked up and as there had been no secretion of any kind for 10 days I left for home, determined to do my best whatever happened.

During the last few days in Houston I had started dionin 5 percent in each eye daily. This I continued for nearly a week. Then all treatment was stopped. I had had 200 mg. cenolate (ascorbic acid) intravenously daily for 17 days. When I arrived in Chicago, February 14th, the acuity was 20/30± and 20/20±, R and L, respectively, with my correction, and this could not be improved by any change in the glasses. The lights were still multitudinous, each one being surrounded by many others, but the halo was not so glary and the single center light or object was more clearly defined. Those who examined my eyes with the slitlamp said they saw that the spots were thinning out—that is, each one was less dense. The progress has continued and now after 10½ weeks, I no longer have halos, my vision with correction is 20/20+ with each eye; the "spots" can still be seen by any keen observer in each of my corneas. I think I can say I have recovered my mental and emotional equilibrium. I can never forget, however, how angry and disgusted I was with myself for the careless moment when, after examining my patient and before washing my hands, I must have fingered my own eyelids.

Entirely from the subjective standpoint I would say cold boric-acid compresses, intravenous vitamin C, and encouragement were the factors I found to be most helpful.

122 South Michigan Avenue.

TEMPORAL ARTERITIS*

Case report with eye findings

Lawrence T. Post, M.D., and T. E. Sanders, M.D.
Saint Louis

Arteritis of the temporal vessels was originally described by Horton, Magath, and Brown[1] of the Mayo Clinic, in 1934. They reported two cases and concluded that this form of a localized arteritis was a previously undescribed entity. The next case of this condition was reported by MacDonald and Moser,[2] in 1937, the further history of whose case is described by the present authors. Three months after the report of this case, Horton and Magath[3] reported a total of nine cases in the literature, seven of them being their own. These cases were so similar, both clinically and pathologically, that they again concluded that this was a new disease entity. This contention has been accepted by most authors, the number of cases now in the literature being approximately 26.[4–14] With few exceptions these cases have been reported in this country, but three were seen in England,[4–5] and one each in Uruguay,[3] France,[6] and Canada.[9]

The disease seems to be limited to elderly persons, the youngest reported being 55 years old. The onset is characterized by headache, which is more or less constant and which tends to be more severe at night, general malaise, weakness, fever, anorexia, and loss of weight. From two to five weeks after onset the temporal arteries become very prominent and tortuous, with inflamed raised nodules along their course. They are very painful and tender to the touch, this condition being present to a lesser extent over the whole scalp. In the early stages pulsations are present, but these disappear as the condition progresses. The temperature usually ranges from 98°-103°F., and the white-blood-cell count from 7,500 to 13,500, with a reduction in the red-blood-cell count to around 3,500,000. Usually there are no other significant physical findings.

The disease generally runs a chronic course of four to six months' duration, the patient being often bedfast for long periods, but never dangerously ill. Pain in the head is usually the most marked complaint. Complete recovery has occurred in every case on record. There is no specific treatment. However, the symptoms in most cases are completely relieved by removal of portions of the temporal artery for biopsy.

The pathologic picture is characteristic and serves to differentiate this condition from periarteritis nodosa and rheumatic arteritis. Pathologically the lesion is a chronic periarteritis and arteritis. There is an infiltration of round cells in the adventitia around the vasa vasorum and in the media of the larger vessels. This infiltration is present to such a marked degree in some areas of the media that complete necrosis with hemorrhage is present. In the areas of destruction of the media it is replaced by a granulomatous type of lesion in which many giant cells are invariably present. The intima in most places is much thickened. This reaction usually reduces the lumen to a marked extent, which in many areas is completely occluded by thrombi. The gross nodular areas represent the cellular infiltration around the vasa vasorum. The etiology is unknown, but the inflammatory appearance of the lesions suggests a low-grade infection. The advanced age suggests that

* From the Department of Ophthalmology, Washington University, and the Oscar Johnson Institute. Read at the seventy-ninth annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, June 10-12, 1943.

degenerative changes in the arterial walls may be a factor.

The following report of a case of temporal arteritis does not represent an addition to the total number in the literature, as this case is the third to be reported, having been described previously by MacDonald and Moser[2] in 1937.

Report of case

The patient, a white housewife, aged 66 years, was examined by Dr. L. D. Thompson of Saint Louis on July 8, 1941, because of failing memory and mental deterioration.

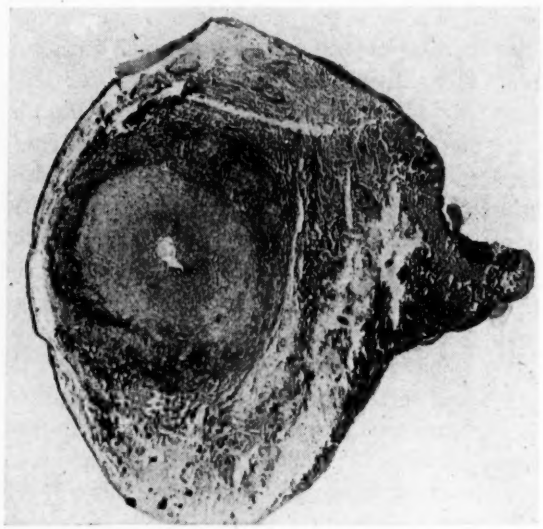


Fig. 1 (MacDonald and Moser). Low-power cross section of temporal artery, showing inflammatory reaction in the media and a marked proliferation of the intima (× 20).

Past history (an abstract of the case as reported in detail by MacDonald and Moser[2]). The patient entered a hospital in Indianapolis on March 1, 1935, because of pain in the temples, fever, sweats, and general malaise. About four weeks before she had noted a tender area in front of the right ear. About a week later the left side of the face became swollen and tender. At this time she became aware that the temporal arteries were prominent, red, and tender. General malaise and weakness were pronounced, and fever and sweats were present every night. Her past history was of no importance in this connection.

Physical examination at this time revealed the left temporal artery to be very prominent from a point just above the tempero-mandibular joint for a distance of about 2½ inches. It was thickened, tortuous, the caliber irregular, and the surrounding tissue hyperemic. Pulsation was present throughout the course of the vessels at the time of the first examination, although this became very much diminished a little later. The right temporal artery was less acutely involved, but pulsation was definitely diminished. The preauricular and postauricular glands were enlarged and tender. Two teeth were found to have periapical abscesses. Both optic-nerve heads appeared normal, and the retinal vessels showed about the usual change for her age. The heart was not enlarged. The blood pressure was 148/90. There was a definite pulsation felt in the suprasternal notch. The remainder of the examination was essentially normal. Except for a slight anemia, the blood examination and serology were normal. Later urinalysis was normal, although a faint trace of albumin and a few casts were noted shortly after admission. X-ray studies of the chest showed the heart to be of normal size.

On March 29th a section of the right temporal artery (three centimeters) was removed for histologic examination. This showed marked hypertrophy of the intima, almost occluding the lumen, with a scarcity of nuclear elements and tendency

to hyalinization (fig. 1). The media showed almost total destruction and replacement by inflammatory cells of fibroblastic variety. Upon the external border of the media in certain zones were huge multinuclear giant cells. (fig. 2). The adventitia was atrophic. Following the removal of this segment, the temperature became normal and remained so until the patient left the hospital one week later. The patient reëntered the hospital on May 13th. She had gained much strength and her general appearance was much improved. She had no fever after the first week. The incision over the right temporal artery was healed. The left temporal artery was smaller and showed no pulsation. It still showed some evidence of inflammation. The eyegrounds were normal. The remaining physical findings and the laboratory examinations were as at first admission. After dental extraction, the patient was discharged.

She was again seen on August 6, 1935, at which time she stated she felt well in every way. The blood pressure was 160/110. The left temporal artery appeared normal, and a normal pulsation was present. At this time there appeared to be an increased narrowing of the temporal vessels and more pulsation in the suprasternal notch. Examination in June, 1936, showed essentially the same findings as the year before.

Interval history. The patient had remained entirely well until two years ago when some mental deterioration was first noted. This had been characterized by memory defect, periods of aphasia, some intermittent depression, and general slowing of mental activity. There had been some tendency to slow progression.

Physical examination (July 8, 1941). The patient was perfectly oriented and rational, but reacted quite slowly. There was a smoothness and lack of wrinkles in the face suggestive of Parkinsonism, but there was no tremor. There were one or two devitalized teeth. The tonsils were barely visible, the remainder of the pharynx being normal. There was some scarring in either temple, but no inflammatory reaction was present. The thyroid gland was not unusual. The right carotid

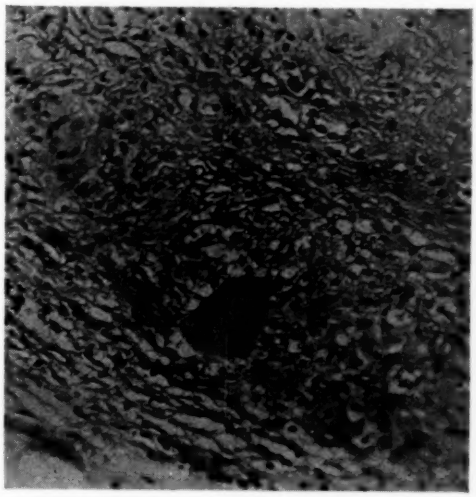


Fig. 2 (MacDonald and Moser). High power through wall of temporal artery, showing chronic granulomatous inflammation in media (× 150).

was definitely dilated. A pulsating mass could be felt extending 2 cm. above the right clavicle, probably an aneurysm of the innominate artery. This was confirmed by fluoroscopic examination, which showed a definite widening of the base of the heart with a pulsating area extending from the right border of the arch into the neck. Over the aneurysm the second heart sound could be heard clearly. Blood pressure was 182/104 in the right arm and 186/112 in the left. There was widening of the heart at the base, particularly toward the right. Sounds, rate, and rhythm were normal except for an accentuation of the second aortic sound. The lungs showed a moderate amount of

emphysema, but were otherwise normal. The examination of the abdomen, pelvis, and extremities revealed nothing unusual.

Laboratory examination. Red-blood-cell count was 4,220,000 and the white-cell-count 7,600. Differential blood count showed segmented forms 54 percent, stab forms 5 percent, lymphocytes 38 percent, and eosinophils, monocytes, and basophils 1 percent each. The fasting blood sugar was 76 mg. percent. The Kahn test was negative. The basal metabolic rate was −14 percent. The urine was acid with a specific gravity of 1.010, and was negative for albumin and sugar. The microscopic examination showed only an occasional white blood cell. X-ray examination of the skull showed a questionable erosion of the floor of the dorsum sellae, and that of the chest a cardiac enlargement with a definite widening of the supracardiac shadow.

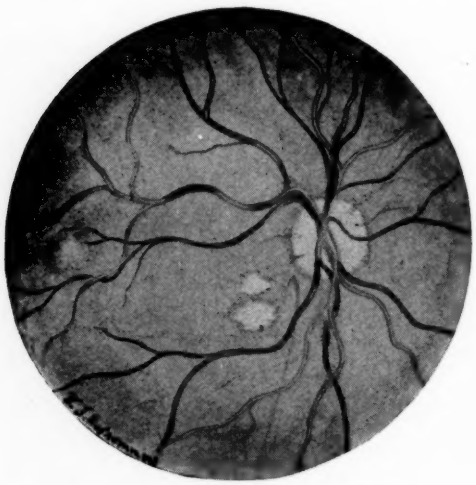


Fig. 3 (Post and Sanders). Fundus drawing, right eye.

The patient was also examined by Dr. A. B. Jones, a neurologist, who reported that the patient was merely an "old woman," older than her years, with no localized findings. He thought the findings were due to cerebral arteriosclerosis.

Ophthalmoscopic examination was done July 9, 1941, by one of us (L.T.P.). The patient had no ocular complaints. The vision was normal, glasses being worn for close work only. The patient's husband was in the hospital because of detached retina, and the ophthalmoscopic examination was done as a matter of interest in view of the past history, and not at the request of the patient, who did not desire an eye examination since she had no eye symptoms. On being informed that the eyegrounds were unusual, she consented to having photographs made of them. The media were clear. The discs were of good color and outline. The arteries showed a marked sclerosis with irregularity of caliber and thickening of the walls approaching the copper-wire stage. In the right eye along the course of the superior temporal artery above the macula were two areas of apparent sheathing, the larger being about one disc diameter in length, the smaller, one half its length, being near the disc. Other similar lesions were noted in the peripheral branches of this artery, and another small area in the superior nasal artery adjacent to the disc. In these places the arterial wall seemed to have undergone a fusiform thickening which was deep red in color. The reflex over these areas was markedly impaired, and the blood column could not be seen. The surrounding tissues showed no reaction nor scarring. Below and nasal to the macula were three confluent areas of silvery exudate. They had indefinite borders, the whole area being about the size of the disc. There was no inflammatory reaction present. No hemorrhages were noted (fig. 3). The left fundus was not unusual except for the sclerosis of the vessels.

On March 28, 1942, it was found that the general condition was somewhat improved on general supportive treatment. She was complaining of some inability to

control the flow of urine. Dr. D. K. Rose found a hyperplastic urethritis which was treated by fulguration in Barnes Hospital on April 3, 1942. At this time the general laboratory findings were essentially as noted previously. The appearance of the fundus of the right eye was essentially unchanged.

The patient died very suddenly of a cerebral hemorrhage on September 12, 1942.

Comment

Although many of the cases of temporal arteritis described in the literature have definite eye findings, very little attention was given this phase of the condition until several months ago, when Johnson, Harley, and Horton[14] reported three cases in which there was marked loss of vision. In their first case a quadrant field defect was present in one eye with nothing in the fundus except a few ischemic areas to account for the loss. In the other eye the vision was reduced to light perception only, with occlusion of some of the upper branches of the central retinal artery and ischemic elevation of the upper margin of the disc. In the second case the vision of the right eye was limited by a corneal leucoma to perception of hand movements. The fundus of this eye was not unusual except for mild arteriosclerosis. In the other eye the vision was completely gone. The optic disc was mildly edematous and slightly pale. From the temporal margin an irregular, whitish, slightly raised region of exudation extended outward about one disc diameter. Several small hemorrhagic extravasations were present along the superior nasal vessels and above the disc. Several days later there was a sudden, complete loss of vision in the right, eye, with no apparent local cause. The patient in the third case had no vision in the right eye and 3/30 in the left. The fundus of the right eye revealed a rather pale disc, mildly edematous, with an area of localized edema of the retina between the disc and macula in which there was a slightly reddened area suggestive of a cherry-red spot. The optic disc of the left eye showed one diopter of edema and the lower temporal quadrant appeared rather pale. The veins were engorged. The authors concluded that in these cases the mode of onset of loss of vision suggested occlusion of the arterial blood supply of the retina or optic nerve. The evidence of occlusion in the retina was insufficient to explain the complete loss of vision, so this must have occurred in vessels too far back to produce fundus lesions.

In the other cases reported, the eye findings are mentioned only incidentally and in relatively little detail. Even so, approximately one third of the cases in the literature show some significant eye lesion. In the series of seven cases reported by Horton and Magath,[8] two cases presented "evidence of phlebitis of one of the retinal veins with hemorrhages and exudates" and one patient had diplopia. In one of the cases reported by Jennings,[4] the left eye was completely blind on admission, having suffered a gradual loss of vision for one week. The disc was pale, with small central hemorrhages, and the retinal arteries were narrow. The right eye was normal at this time, but within six days a similar picture developed in this eye with complete loss of vision. The diagnosis of the eye consultant was bilateral obstruction of the retinal arteries. Bain[5] stated that in his case there were severe photophobia and bilateral "peripapillary atrophy." Both of the cases reported by Dick and Freeman[7] presented blurred vision. In their first case, the vision of the right eye had been poor since birth. During the course of the disease "the vision in the left eye became blurred. The optic disc was twice the normal size

and was slightly edematous. A small hemorrhage was present on the disc. The visual field was reduced to a small point." There was no statement as to the final visual result. Their second patient complained of soreness of the eyes and blurred vision with diplopia for one week. Examination showed "tortuous retinal arteries, sluggish extraocular movements, and poor vision in the right eye." In the case reported by Scott and Maxwell,[10] the fundi were normal at the onset. The patient was also found to have a diabetes with a high renal threshold. About two months later she was complaining of loss of vision in each eye, more in the right. The fundus of this eye could not be seen because of opacity in the lens, whereas the left showed no change in the arteries, but numerous small patches of exudate were seen in the peripapillary area.

In the experience of both authors of this report the fundus picture in this patient seemed unique. To our knowledge no similar arterial lesion has been previously described either in temporal arteritis or in any other vascular condition. The nearest picture approaching the appearance of this fundus was seen in plate 56 in the Wilmer Atlas. The plate is entitled "retinal arteriosclerosis," but from the description there had been a marked loss of vision apparently from an occlusion of the arterial blood supply occurring at the time of a cerebral hemorrhage. In another plate of the same eye (no. 26), made three months later, the localized arterial lesions had disappeared, but a marked generalized attenuation of the retinal vessels was present with glaucoma.

The exact nature of the localized arterial lesion seen in our patient presents a problem in interpretation. It does not seem to be an area of acute inflammatory arteritis related directly to the lesion in the temporal artery, as it did not take place for some months after the temporal lesion occurred. Also there was no evidence of periarteritis or reaction in the surrounding tissues, as would have been expected in the case of an acute inflammatory lesion, the changes here being localized in the wall of the artery. From the appearance of the fundus alone, the best conclusion is that arterial changes are on a degenerative basis, but this does not explain the brilliant-red color of the localized areas. Whether inflammatory or degenerative, the lesion at this stage must have been relatively inactive as the appearance of the arteries was unchanged after eight months. The area of exudate could be an infarct caused by an occlusion of a small branch of a retinal artery. It is obvious that the arterial degenerative changes are not restricted to the retina, for the patient had an aneurysm of the innominate artery and had marked cerebral sclerosis, dying of a cerebral hemorrhage. It is possible that the whole fundus picture could be explained on the basis of the patient's arteriosclerosis and hypertension, although the appearance of the local lesions is unique.

Despite the fact that eye lesions are very commonly seen accompanying temporal arteritis, we believe that no particular one is characteristic. The two commonly seen are occlusions of the central retinal artery or larger arteries, and a localized arterial lesion with hemorrhages or exudates not unlike those seen in certain cases of arteriosclerosis. It is also possible that certain fundus changes diagnosed as arteriosclerotic might be degenerative similar to those seen in this patient.

We also agree with the statement made by many other authors that the arterial disease is not confined to the temporal and ophthalmic vessels, but is scattered extensively throughout the vessels of the body, especially those of the head and neck. We are inclined to believe that the

condition is fundamentally a degenerative one, which at times undergoes a localized acute inflammatory phase, this being particularly prone to occur in the temporal vessels. The fundus picture in our case may represent one stage of inflammation in a retinal artery.

640 South Kingshighway.

References

[1] Horton, B. T., Magath, T. B., and Brown, G. E. Arteritis of the temporal vessels: A previously undescribed form. Arch. Int. Med., 1934, v. 53, March, pp. 400-409.

[2] MacDonald, J. A., and Moser, R. H. Periarteritis nodosa and arteritis of the temporal vessels: A case report. Ann. Int. Med., 1937, v. 10, May, pp. 1721-1726.

[3] Horton, B. T., and Magath, T. B. Arteritis of the temporal vessels: Report of seven cases. Proc. Staff Meet. Mayo Clinic, 1937, v. 12, Sept. 1, pp. 548-553.

[4] Jennings, G. H. Arteritis of the temporal vessels. Lancet, 1938, v. 1, Feb. 19, p. 424.

[5] Bain, C. W. C. Arteritis of the temporal arteries. Lancet, 1938, v. 1, Feb. 26, p. 517.

[6] Theobriand, M. Syndrome neuralgique, cranio-fronto-temporal, d'origine sympathétique: Resection de l'artère temporale superficielle. Bull. et Mem. de la Soc. des Chir. de Paris, 1939, v. 31, p. 136.

[7] Dick, G. F., and Freeman, G. Temporal arteritis. Jour. Amer. Med. Assoc., 1940, v. 114, Feb. 24, pp. 645-647.

[8] Bowers, J. M. Arteritis of the temporal vessels. Arch. Int. Med., 1940, v. 66, Aug. pp. 384-392.

[9] Sprague, P. H., and MacKenzie, W. C. A case of temporal arteritis (Horton-Magath syndrome). Can. Med. Assoc. Jour., 1940, v. 43, Dec., pp. 562-564.

[10] Scott, T., and Maxwell, E. S. Temporal arteritis: A case report. Intern. Clin., 1941, v. 2, June, pp. 220-223.

[11] Hoyt, L. H., Perera, C. A., and Kauvar, A. J. Temporal arteritis. New Eng. Jour. Med., 1941, v. 225, Aug. 21, pp. 283-286.

[12] Schaefer, C. L., and Sanders, C. E. Temporal arteritis. Amer. Heart Jour., 1942, v. 24, Sept., pp. 410-411.

[13] Murphy, J. R. The syndrome of temporal arteritis. New York State Jour. Med., 1942, v. 42, Dec. 1, pp. 2236-2237.

[14] Johnson, R. H., Harley, R. D., and Horton, B. T. Arteritis of the temporal vessels associated with loss of vision: Report of three cases. Amer. Jour. Ophth., 1943, v. 26, Feb., pp. 147-151.

MILITARY OPHTHALMOLOGY*

WILLIAM THORNWALL DAVIS, M.D.
Washington, D.C.

In the Military Surgical Manual of the National Research Council for Ophthalmology and Otolaryngology,[1] the Surgeons General of the Army and of the Navy set forth a fact that is not yet fully grasped by the profession at large. They have not strongly emphasized the fact that the overspecialization which has taken place since the last war cannot be carried over into the military and naval services. They have, through the medical departments of the Army and of the Navy, placed the ophthalmologists in their special work to a remarkable degree. Speaking as a veteran of two major and several minor wars and having no inhibitions and no reason to tone down the hard necessities, I must tell you that the services need doctors, great numbers of them. When a major engagement takes place, all the doctors in the world would not be sufficient.

The need for experienced specialists is largely confined to base hospitals and hospitals in the home country, also to the large Army Posts and Cantonments. It is clear, therefore, that, compared to the total number of doctors in the service, there is need for relatively few ophthalmologists. It must be obvious also, that many ophthalmologists will have, of necessity, to become Army surgeons, in any field, no matter how far removed from their specialty. Our specialization has become too narrow. The use of ophthalmologists for purposes other than ophthalmology is not purposely done by the Army and Navy. It is a military necessity.

The stern necessity of war has reduced the previous physical standards including those for eyes. Before the attack on Pearl Harbor by the enemy, our standards were those of peace time, when physical perfection was more or less required. We had a great population from which to draw and a pitifully small Army that had been neglected and starved of necessities in the matter of modern equipment. There was, as has ever been in our history, a neglect of preparation for the inevitable. The result now, as always, is terrific waste of lives and material and money. The hurried building of enormous fighting forces on land and sea necessarily results in confusion and waste in time, effort, and the lives of our sons and resources. The pacifists, the "sob sisters," the spirit of "I did not raise my boy to be a soldier," some of the educational institutions that exercised very considerable antimilitary and antipreparation-for-war influence, have done us irreparable injury, not to speak of the lesser influences such as birth control and alien peoples among us who have quietly, skillfully, and continuously undermined our morale; all enemies of the Republic, dangerous and destructive. Thanks be to Almighty God, such influences are being successfully combated; for example, by the introduction of some control in the educational institutions whereby physical education will be enforced on the student body, something that has been almost entirely neglected.

And what has this to do with ophthalmology? Much, very much; if you would have good eyes, without which a man is so seriously handicapped in the armed forces as well as in civilian life but to a lesser extent, those eyes must be in a strong and vigorous body. Such vigor can result only from physical training of the whole student body from the first

* From the Department of Ophthalmology, The George Washington University School of Medicine. Read before the New York Academy of Medicine, January 18, 1943.

grade in school to the end of the senior year in college—not from specialized training given to a few superathletes to make a team with which to advertise the university at the physical expense of the team and, worse, of the student body. A shortsighted and selfish attitude indeed.

We may not like the arbitrary methods of the military, but this very moment it is those methods that are saving this country from being overrun by savage fiends, infinitely worse than the hordes under Genghis Kahn who overran and destroyed the civilization of that day. We should be thankful, indeed, for the wisdom, foresight, and patriotism of those who established the military and naval academies, which are not excelled in the world, to give us young men trained thoroughly, mentally and physically, trained in patriotism first and thought of self afterward. Would to God our educational leaders had taken a page from history and had had the good sense to apply it to their institutions years ago. What a different situation would have presented itself now! There would have been many tens of thousands of stout fellows, well educated and disciplined; thoroughly alive to military necessity through study of the military history of our country. The educational institutions of our country have been sadly lacking in the preparation of the youth entrusted to their charge for the greatest of all tests that comes at least every generation. Because of this, certainly in part, many have been rendered physically unfit for military duty because of weak and undeveloped bodies, often with resulting defective eyes. It is our duty as members of the medical profession to combat these evil influences; it is our duty as ophthalmologists to combat the too-long hours in hot school rooms, too little physical training, and too much home work. The young people have insufficient time for out-of-doors exercise and proper hours of sleep during their adolescence, the time they need it most. This is preventive military ophthalmology, and we should work for it and fight for it. The first graders who are suffering such insults from the schools must be the fighting men of tomorrow. Have we a greater duty than to do our best to abolish our system of faulty education and erect a sounder one in which the development of the body takes equal precedent with the development of the mind?

In speaking of delayed mustard-gas lesions, Philips[2] describes patients as saying that they had been symptom-free for 10 to 20 years, had continued work, and had apparently suffered no visual disability. From the onset of the delayed keratitis they experienced rapid deterioration of sight.

From a somewhat limited experience in treating eyes that have been damaged by strong alkalis and acids, it would appear that the course of disease in eyes with severe mustard-gas burns follows a rather close parallel: slow degeneration ending in phthisis bulbi. There may or may not be pain, although pain is likely to be suffered at intervals.

Dr. Ida Mann[3] states that the clinical pathology in mustard-gas lesions in man and in rabbits is about the same. Since the life of a rabbit averages 10 years, she believes she can calculate the course of the disease following mustard burns closely in man as to time of the various developments ending in destruction of the eye. This, of course, takes into consideration the relative expectancy of life of the two animals, rabbit and man. The conclusions are as follows:

1. The clinical pathology of the lesion involving the limbus is similar in man and the rabbit.

2. The reaction in man and the rabbit is dependent on the size of the dose and the anatomic situation. The effects of

TABLE 2*

COURSE OF MUSTARD-GAS LESIONS IN MAN, COMPILED FROM OBSERVATIONS ON 65 CASES

Exposure to mustard gas—no immediate sensation

↓

Burning sensation
Hyperemia and lacrimation in 2 to 6 hours

↓

Very mild cases	Mild cases	Severe cases
Lids not closed. Conjunctiva hyperemic. Cornea normal in 24 hours	Injury to epithelium. Punctate staining. Hyperemia. Edema of lids and epithelium. *Eyes partially closed*, 3 to 4 days	Hyperemia of conjunctiva. *Edema of epithelium and substantia propria. Damage to limbal blood supply.* Edema of lids, and eyes closed at 8 hours
↓	↓	↓
Clears up within a week	Clears up in 4 days to six weeks	Very Severe Cases
	↓	↓
	Healed corneal lesion. *Will not relapse*	Eyes open at 5 days
		↓
		Subsidence of primary edema and beginning *subconjunctival hemorrhages* 3 to 5 weeks
		↓
		Vascularization and secondary edema—8 weeks or more
		↓
		Subsidence. *Probably will not relapse*

Eyes closed for 7 weeks	
↓	
Persistence of edema	
↓	
Increased and repeated vascularization. 2 months to 1 year	
↓	
Subsidence with vascular scar. Unsafe. May break down 1 to 6 years	Cholesterin and fatty degeneration
	↓
	Recurrent ulceration 6 to 20 years

Perforation
↓
Phthisis bulbi

vapor and liquid are different only when the actual amount of mustard gas that soaks into the tissues is different; that is, the difference is quantitative only.

3. The late keratitis in man is seen as a degenerative ulceration depending on the initial damage sustained by the limbus and cornea, and not on any continued action of mustard gas or any of its breakdown products.

4. From conclusion 3, it follows that, in the present state of our knowledge, prophylaxis is likely to be much more important than treatment. It is very difficult to assess the value of treatment in man, since the dose of mustard gas received can never be known and no two cases will be comparable. Two eyes of the same patient may be comparable, though by no means is this always the case, even when the lesions are due to vapor.

EYE INJURIES

Love[4] states in a personal communication that since our troops have engaged in only a limited amount of combat and since the statistical material has not been compiled, little information is available at this time regarding combat injuries of the eyes in the Army.

* From Mann and Pullinger. Amer. Jour. Ophth., 1943, v. 26, p. 1276.

With reference to the prophylactic measures, it was recognized early in the emergency that protective goggles were necessary for the armed forces. Consequently, a type was adopted and since that time has been furnished to all the troops in armored divisions.

About December, 1941, the War Department adopted the policy of furnishing spectacles to all military personnel that required their use. To facilitate the furnishing of spectacles and the replacements for them in the theater of operations, mobile optical units have been organized.

Steps have also been taken to provide corrective lenses for the gas mask for such troops as require them.

Lenses of plastic are being developed.

In a personal communication Matthews[5] says that ophthalmic injuries in the Franco-German War were 1.18 percent of all wounds. In the Russo-Japanese War they were 2.2 percent. In World War I they were 5.2 percent. The percentage in this war is exceeding this figure.

Duthie[6] observes that (1) the injuries to the eyes have, generally speaking, shown the same characteristics as those in civil practice; (2) they are mostly extremely severe types of injury; and (3) the advances made in the general treatment of casualties have shown that it is unwise to think that recovery of such severe injuries is unlikely. Meticulous treatment is indicated.

Injuries to the orbit

When a foreign body in the orbit is giving no trouble it is wise to leave it alone. The disfiguring injuries of the face are largely in the hands of the plastic surgeon, but ocular symptoms, particularly diplopia, are of significance, as they usually indicate a more severe type of lesion. The prognosis depends on early diagnosis and early manipulative treatment. Collaboration of the plastic surgeon with the ophthalmic surgeon is of great value.

Lids

Every type of laceration may be seen; severe burns are the most important problem. "Tanning" does not give good results with the lids, as it is too rigid and causes too much contracture. It is better to use the gentian-violet silver-nitrate technique. Skillful nursing is necessary to care for the conjunctival discharge that frequently complicates such cases. Stellard's conjunctivo-dacryocystotomy is of much value. Early plastic repair of the lids is of great advantage and should be done where the military situation permits.

Burns

Thermal burns are far greater in this war than ever before. They accounted for 60 percent of all casualties at Pearl Harbor. The principal factors are, according to Matthews,[5] the ignition of oil or gasoline aboard ship; the use of highly volatile gasoline for all purposes; and the incendiary bomb and the flame thrower. The face and the hands are mostly involved. At Pearl Harbor many of the men on our ships had on only trousers. They had burns from the waist up, the face and hands and the feet, as the latter were bare. "Aviator's burn" is the term given to flash burns of the face and hands from the explosion of gasoline vapor in the cockpit. Diminution of the burns of the eyes might be avoided by the wearing of goggles. Personally, I know from service in combat with soldiers, marines, and sailors that it would be most unlikely that they would have them on at such a time and there is no time to put them on when combat starts.

The antiflash gear (asbestos gauntlets and hood) is used successfully. Matthews[5]

remarks that the men object to wearing it in the tropics. I would deem it almost impossible to wear such things in combat in the equatorial heat.

Gifford[7] estimates the extent of corneal burns by noting the transparency of the cornea and the intensity of the corneal staining with fluorescein.

General, widespread burns may coexist with the eye lesions, and the ophthalmologist should be fully informed of the procedures used in such situations.

For burns of the lids Wakeley[8] recommends the use of 1-percent gentian-violet jelly with merthiolate; aboard British vessels it is immediately available and easily applied in combat. Suturing the lid margins is of value in assisting in the prevention of contracture and also in corneal injuries at the front. Powdered sulfanilamide becomes caked; the granulated form less so, and hence, it is better. Mixtures of sulfanilamide and mineral oil were used at Pearl Harbor with satisfaction. A 5-percent aqueous emulsion without bandaging is recommended by Matthews.[5]

An interesting fact is that sailors immersed in the sea for hours after torpedo attacks with burns, show the wounds to be clean and to heal satisfactorily. This I found to be a fact in the Moro campaigns in Mindanao. This was jungle fighting such as we are again having in those beautiful tropical isles. Our soldiers suffered from terrible knife wounds when we would be many days' or weeks' march from the coast. The wounded men would have to be carried on litters made of bamboo and rattan by the natives. Proper care could not be given; the wounds would often become infested with maggots. Upon their return to the base hospital or even to the beach I would wash these wounds in sea water; not boiled but taken from the ocean in a sterile container if one was available; often I would place the patient in the sea so that the gentle waves would wash over him and then allow him to lie in the sun on the beach to dry. These wounds would be clean, and if it were possible to have aseptic sutures they would heal by first intention. I think sea water from clean oceans in the tropics has a valuable aseptic effect. It also is soothing, cleansing, and is isotonic with the blood serum.

Foreign bodies

Matthews mentions the great differences in foreign bodies in the globe from World Wars I and II. So far in this war there is a greater incidence of nonmagnetic metallic fragments due to the substitution of aluminum and magnesium alloys for brass and steel in the manufacture of bomb and shell cases. These fragments become encrusted after several days and do no harm. Ophthalmoscopically, they may resemble synchysis scintillans when they are very small and numerous.

In one series, in the Polish war, about 4 percent of foreign bodies were in the posterior segment of the globe. The typical signs were a deepened anterior chamber, depression of the iris, folds in Descemet's membrane, intraocular hemorrhage, and subsequent retinal detachment; all of the eyes with this involvement were lost.

In World War I shell fragments were the most frequent causative agents. Now with the use of the aerial high-explosive bomb, flying débris is the chief cause. In cities, glass is the most important; such foreign bodies are difficult to localize. Falling shrapnel of friendly anti-aircraft fire also accounts for many casualties. Lagrange (quoted by Matthews) analyzed the orbital and ocular wounds of the last war with the following observations. Missiles passing above the orbit, injuring the frontal bone, produced fractures in the region of the optic foramen and superior orbital fissures, causing disorders of the motor and optic nerves without injury

to the eyeball itself. Missiles passing below the orbit often produce retinal injuries by concussion, usually affecting the macular region. Where any of the orbital walls are crushed, concussion injuries of the globe are likely to occur with chorioretinal laceration and subsequent retinitis proliferans and retinal detachment. Such detachments are rarely amenable to surgery. They differ from those detachments due to recurrent retinal hemorrhage. Proliferation stems from the ruptured choroid in the injury cases. Doherty, as quoted by Matthews, noted that unusual conditions obtained in war are: (1) spastic blepharospasm and entropion caused by irritation of the infraorbital nerve, usually from a retained intraorbital foreign body near the apex; (2) avulsion of the optic nerve; (3) enophthalmos due to depression of the wall of the orbit with consequent enlargement of the orbital cavity; and (4) hematic ring of the disc, a sequela of hematoma of the sheaths of the optic nerve.

In military surgery the size of a foreign body is likely to be larger; it is less likely to be magnetic, the period of time before treatment can be instituted is longer and sepsis is more likely to occur. It is obvious that the prognosis is worse in war injuries. It is well to put the eye to the magnet even though the X-ray picture is negative, since more than one foreign body may be within the globe. My personal experience with foreign bodies in the globe has not been a happy one. Most of the eyes are eventually lost from detached retinas, secondary glaucoma, chronic uveitis, complicated cataract, and other distressing sequelae not to mention the possibility of sympathetic ophthalmia. Many of these complications occur very late, several or more years after the injury. Removal of the foreign body may be accomplished, but even though accomplished the end results are often disastrous; except for iron and copper it may be best to let them remain.

Sympathetic ophthalmia

The risks are much greater if prolapsed and incarcerated uvea is dealt with after some time rather than within the first 48 hours. This is of the greatest importance. Eyes that have had such a type of injury and that come under observation a fortnight afterward should be enucleated if the eye looks sick and has a low-grade uveitis, is soft and with ciliary injection.

Duthie[6] thinks eye work in general hospital suffers, being placed in a secondary position, with which we all agree. He thinks, and I am sure all ophthalmologists will agree, that injured eyes should be in an ophthalmic unit in a general hospital.

In the treatment of eye wounds in combat, which includes air raids and blackouts, first-aid workers and medical personnel are instructed to anesthetize the eye and apply sterile dressings, leaving definitive treatment to more expert ophthalmologists if the surroundings and personnel are conducive to such care.

The differences are reiterated for emphasis: Enforced delays in handling war casualties, as before mentioned, make wound contamination obviously more likely to occur. The coexistence of shock and other serious injuries may place the eye injury in the position of secondary importance. There may be inadequate or no facilities in the field. The last named can scarcely be realized or visualized by those who have not been in combat.

Our military personnel are instructed to observe the following routine in cases of perforating and other severe wounds in the field and combat.

1. Anesthetize the eye with 1-percent butyn or 0.5-percent pontocaine.

2. Gently remove any superficial foreign bodies with cotton-wound applicator moistened with boric-acid solution.

3. Flush the eye with boric-acid solution.

4. Close the eyelids and apply a fairly firm dressing, held in place by adhesive tape.

5. Evacuate as rapidly as possible, so that the patient may receive proper ophthalmologic care promptly.

6. Do not close the wound with sutures.

7. Do not excise any tissue protruding from the wound.

8. Do not cover the eye with a conjunctival flap, unless specifically equipped to do so.

Two German writers are said to have successfully sutured corneal wounds with human hair. They claim better optical results than with a conjunctival flap.

When an eye is destroyed, and bits of uveal tissue are buried in the surrounding tissues, they should be sought for and removed to prevent sympathetic ophthalmia. Early probing in the orbit for bullets or shell fragments is unwise. Infection may be introduced into the orbit or through skull fractures to the meninges.

Grossly lacerated lids may be sutured over the cornea to prevent drying of the latter. Walker, quoted by Matthews, introduced sulfanilamide crystals directly into the anterior chamber in severe lacerations of the cornea at Pearl Harbor. This was done before closure with a conjunctival flap. The results were gratifying. All army personnel carry into combat packets of sulfanilamide on their persons and are instructed in its use.

Cornea

Multiple foreign bodies are the most important and the most frequent injury. Subconjunctival foreign bodies had best be let alone. They usually work to the surface; if not, they rarely cause trouble.

If the eye is quiescent when there are numerous foreign bodies in the cornea, it is best to let it alone rather than attempt complicated or even simple methods of removal, since no irritation is present. Subsequent sepsis is rare. Even if the eye is considerably injected as the result of the explosion of an incendiary bomb, for example, too much interference is probably unwise.

Concussion

Concussion is frequent and is the result of (1) direct blows on the eye, (2) blows on the bones of the face or orbit, (3) foreign bodies traversing the orbit, and (4) blast.

These were the most frequent injuries of the last war as of this one, and there is voluminous literature concerning the subject.

The various conditions arising from such injuries are iridodialysis or, conversely, rupturing or tearing of the pupillary margin; concussion cataract; all types of intraocular hemorrhages; retinal-concussion changes characterized by large areas of dark hemorrhages; ruptures of the choroid, concentric to the disc, or, more occasionally, a tear radiating from it, and avulsion of the optic nerve.

Intraocular hemorrhages are important; if they are anterior and persistent, secondary glaucoma may supervene. Paracentesis of the anterior chamber is in order. Posterior hemorrhages are sinister. Efforts should be made at once to favor absorption and delay coagulation, thus preventing the formation of solid masses in the vitreous which resist absorption. They may become organized.

A solid clot may cause false light projection although there be no lesion of the retina or choroid. This may lead to the enucleation of an eye that might have the possibilities of restoration of vision.

The patient, according to Duthie,[8] should *not* be kept in bed but should move about. After a considerable period, if ab-

sorption of the clot is not taking place, scleral puncture may be considered. Subconjunctival injections of normal saline may be tried.

RUPTURE OF THE GLOBE

The majority of cases are caused by flying splinters and glass. Many globes are completely destroyed; many hopelessly injured. Personally, I think we should make a careful inspection and a more careful decision where there seems to be even a faint hope of saving the eye.

There may be rupture by contrecoup or rupture caused by a foreign body penetrating into the orbit but not the globe and causing rupture by striking the eye itself. If the uvea is extruded it should be cut off. In cases in which there are severe wounds in the ciliary region, in unilateral cases, and in those presenting other intraocular complications, Duthie advises enucleation. This is good advice when we consider the possibility of sympathetic ophthalmia. Nevertheless, I think we should be conservative *where military conditions permit.*

Small conjunctival flaps sutured firmly to the episclera I consider to be better than large ones and also better than scleral suture.

Like myself, Duthie is conservative in the use of sulfa drugs in cases of intraocular sepsis or the prevention thereof. All other aseptic and antiseptic precautions should be followed. The end results of ruptured globes are most frequently very sad in military as in civil practice, and this is the experience of us all. They are likely to be sadder in military medicine than in civil practice for obvious reasons.

It appears best, in the combat elements of the army active in the field, to bandage the injured eye and get the man back to the nearest place where adequate treatment or enucleation can be carried out. This applies to all injuries of the eye of any moment, except gas injuries, where bandaging is contraindicated. Obviously, the treatment of more than trivial injuries cannot be adequately carried out in a fast-moving combat force. It would seem that the wounded and injured, under present combat conditions, may have to wait long and travel far before they may receive adequate treatment. In the days of marching armies, wagon trains, and no air strafing, the field hospitals were relatively easy of access. Today they may be hundreds of miles away and accessible only by air or rapid motor transportation. There may be no such transportation available for the wounded. Combat calls for everything the army has when a battle is on. The regulations state that so much transportation will be available for the medical department. This is under the direct command of the ranking general, not the medical officer, and he has to win the battle or all are lost, wounded included. Under such conditions the theoretic transportation may be unavailable for a time; hence, it is every man to his job to do the best he can under the worst possible conditions, lack of food, water, shelter, and supplies.

In a personal communication from Dr. Meyer Wiener,[9] honorary consultant in ophthalmology to the Navy, he says:

"I think one of the striking features about foreign-body injuries in this war is the fact that we have not had a single one that was magnetic. Even when they were removed, and looked like iron or steel, they failed to be attracted by the magnet. An analysis of quite a few proved them to contain a large amount of copper. Then we have had a number that looked like aluminum and proved to be such when we had an opportunity to test them. We make no effort to remove aluminum, as it has proved to be harmless, thus far. We had one officer who had multiple fragments of aluminum all over his body, including some in the knee joint, which had to be removed. These were sustained while in action on a destroyer on August 7th during the first battle for the Solomons. One penetrated the left eye. You could see the tract through the cornea, hole in the

iris, tract through the lens capsule and the foreign body in the lens, just above dead center. We left it in. The patient has had no irritation whatsoever and his vision in that eye today is 20/20—1.

There is another boy from the Solomons who had one eye shot out, with multiple fragments in the right eye, some opacity in the lens, bands in the vitreous, and much blood. We have also treated that expectantly, and his vision has gradually crept up from bare perception of light with poor projection, to ability to count fingers at 6 inches and increasingly improving light projection, tested with the lids closed.

As in the last war, we have seen injuries to the retina and rupture of the choroid from concussion, without any visible external involvement. One case was seen recently with a double rupture of the choroid. The worst cases are the ones where the eye has been penetrated by large objects, especially in the ciliary region. Most of these have had to be enucleated. There have been a number of lid injuries which have required plastic repair. Thus far, we have not had to reconstruct any by pedicle grafts. Dr. Lucic got a beautiful result in a case where the lower half of the right upper lid had been burned away by molten metal, by transferring a segment from the opposite eye, including a thin margin from the brow, and reversing it so as to make a row of lashes. I acted as his first assistant. You may recall him as he was at Wilmer Institute for several years before coming here. I think it should be stressed that the all-important thing is to give adequate proper immediate care, which would obviate the necessity of doing many plastic repairs. We had one recently which demonstrated that. We worked on it for over two hours but finally did get a most satisfactory permanent result. Of course, one cannot always do this under actual battle conditions; but it is my belief that if they do not have the time or conveniences, they should clean up things as best they can, do no débridement, and let the fragments fall as they will. They can then be separated later and readjusted. This is naturally only second best.

I think that you should bring up the subject of malingering. Perhaps some of these fellows are not really trying to gyp the Government, but are shell shocked. At any rate, they must be considered, as we have a lot of them. They are easy enough to catch when only one eye is involved; but when it is claimed that both are affected and equally so, it is a different matter. We have one now, who I am convinced is a fakir but feel that he has been so well coached that we are going to have a hard time proving it.

I thought you would be interested in one case we had of arteriovenous aneurysm of the lateral sinus, with marked proptosis, dilatation, and tortuosity of the vessels, both externally and retinal. Dr. Leonard Furlow tied off the internal carotid, and the swelling is about gone, with almost normal vision. This case was due to concussion but there was no external injury."

Major W. Randolph Lovelace, II,[10] at Wright Field, in a personal communication says:

"The latest work that is being carried on in the Air Corps is that on night vision, color vision, depth perception, and the approaching possibilities of revamping visual requirements for pilots to the lowest limits, commensurate with good performance, rather than the highest possible limits. As far as this laboratory is concerned, I am sure it would be all right to mention the fact that an apparatus is being developed to determine accurately and quickly the degree of night vision present in all flying personnel and that oxygen equipment is being designed so that it can be comfortably used in conjunction with Air Corps goggles. Color vision, of course, is important from the standpoint of detecting camouflage and from the standpoint of night operations."

The different branches of the service naturally have different eye requirements. Those of the Air Force are the most severe, as one would imagine. Incidentally, this leads on to this interesting corollary; there are many investigations and original research problems as well as the results of practical experience which are adding a great deal to our knowledge of the visual act. It is not proper to speak of many facts which have been discovered but they pertain to color vision, night vision, anoxia, and high-altitude flying, and the results on the visual act as to visual acuity both central and peripheral, and disturbances of ocular motility. Much study has been done on dark adaptation. Its importance lies in the necessity for keeping the eyes of the men in the fighting air force dark adapted all the time when they are on duty. It appears that by having them wear red glasses, the dark adaptation is not interfered with and they can see sufficiently well to read while waiting for the call to take off. I am informed that

illumination of the cockpit by red light is better than the "black light"; that is, ultraviolet light acting upon fluorescent dials on the instrument boards.

Dark adaptation and night blindness have received much attention and practical study since the beginning of the war. Much of practical value has been determined. Knowledge of dark adaptation, light sense, and light differences are of great practical use even though the laboratory methods are not satisfactory. Attention is directed to the Military Surgical Manual,[11] where the writer speaks of night blindness and uses the term "war hemeralopia" as applied to soldiers. This did not occur in our Army but did in the armies of Europe who were half starved, as was the whole population, as it is again today. It is, of course, due to avitaminosis A and is curable by the exhibition of this principle when it is available. This manual also deals with diseased eyes with poor night vision and also malingerers and neurotics. Those with diseased eyes would, of course, be quickly discovered and eliminated. Malingerers are subject to court martial and severe punishment, and neurotics do not long exist in a military force. My long military experience taught me this. Malingerers do not last very long when they get into the hands of their comrades, who know full well how to handle them. Incidentally, a good stiff top sergeant is the best cure I know for a neurotic.

Factors affecting night visibility[12] (Military Surgical Manual[11])

A. Purpose

For night operations it is essential that troops be trained to avail themselves of all existing means whereby night vision and the ability to observe are improved. All individuals do not adapt their eyes to darkness at the same rate of speed. It is therefore desirable that commanders know what persons within their organizations have faulty night vision. The ability to see well in the dark is improved by systematic training.

B. Factors influencing development of dark adaptation

1. The time necessary for dark adaptation can be shortened by avoiding bright lights prior to going into darkness.

2. Lack of oxygen markedly decreases the ability of the eye to adapt to darkness. At an altitude of 16,000 feet this reduction is such as to require 10 times as much light to see an object as would be required by the same individual at ground level. Even at 4,000 feet, impairment of night vision is noticeable. Crews of night fighters who are climbing fast should take oxygen from ground level if they wish to make the best possible use of their vision on dark nights. More oxygen than that normally required will not further increase night vision.

3. Persons suffering from a severe lack of essential food factors (vitamins) will show a diminution in their ability to adapt to darkness. In these cases, resumption of an adequate diet or the feeding of vitamins A and D will return the night vision to normal, but an excess of these vitamins will not further improve night vision.

C. Factors interfering with maintenance of dark adaptation

Eyes adapted to darkness will remain so only in the absence of interfering lights. Such a condition, however, seldom pertains in warfare. Light interference may be of many sorts: dash lights, searchlights, gun flashes, flares, and reflected moonlight. Individuals must be made to realize the necessity of avoiding the effects of such factors. Conditions in the combat zone usually dictate that motor movement be made without lights. When

lights are allowed, drivers of motor vehicles should be especially instructed to keep lights within the vehicle at a minimum and to take only fleeting glances at instruments.

D. Other factors affecting night visibility

1. Any form of distraction will result in a less efficient observer. Outstanding among these are the discomforts occasioned by cold, noise, and objects interfering with the field of vision. Fatigue tends to inattention and greatly interferes with night observation.

2. Reducing the contrast between an object and its background detracts from night vision. Anything which scatters some of the light, such as a scratched or dirty windshield, has this effect.

3. In dim lights a large object may be visible when a small one is not. For this reason, night binoculars, designed to admit as much light as possible, help night vision by increasing the apparent size of the object. The use of night glasses is restricted by the fact that they limit to some degree the quantity of available light rays reaching the eye, restrict the field of vision, and must be held without vibration.

E. Training in proper use of eyes

1. In daylight objects are seen most clearly with the center of the eye but at night this ability to see images most sharply shifts to a position slightly off center. For this reason, during darkness the gaze should be directed about 10 degrees to one side of the field that is being observed.

2. The eye is also subject to fatigue. An object will be most distinct immediately upon looking at it. If, however, the gaze remains fixed upon the particular object it gradually loses its distinct outline and blends with the surroundings.

3. Night observers should be taught to employ a roving movement of the eyes in a series of short jumps. Nothing can be seen while the eyes are actually in motion, but vision is best just after they have been moved. This fact is of special significance to the air observer. Rapid changes of angle of bank, producing this effect of rapid motion of the eyes may result in failure of a pilot flying a night mission to see an object on one side of his line of flight.

F. Test for night vision

1. The following rough measurement of night vision may be made by testing men outdoors on a dark night before the moon has appeared. A night will be considered too bright for test purposes if, after 25 minutes of dark adaptation, individuals can read small newspaper type without the assistance of light.

2. Mount on a post at the end of a level space 5 yards wide and 40 yards long and away from all artificial light, a black target on which is a movable white strip 6 by 24 inches. Mark off distances from the target at 5-yard intervals. After the person to be tested has been kept away from all artificial illumination for 25 minutes, direct him to walk slowly from the far end of the test area toward the target. The distance at which he is first able to identify correctly the position of the white strip is noted. While the person being tested is walking back for another trial, the position of the white strip is changed. Several trials are made. Correct recognition should take place at an average distance of not less than 20 yards from the target.

G. Summary

1. Instruction designed to improve the ability to observe in darkness will include training in the proper use of the eyes at night and a consideration of those factors assisting or interfering with night visi-

bility. The following points will be especially stressed:

a. The importance of and factors governing pre-adaptation.
b. Proper methods of search and the employment of "off center" vision.
c. The detrimental effects of night adaptation of: (1) lack of oxygen at high altitudes; (2) light on the dark-adapted eye; (3) distraction, with special reference to cold, noise, and objects interfering with the field of vision; (4) fatigue; (5) light dispersion, emphasizing the necessity for scrupulously clean windshields.

2. Unit commanders will identify those individuals within their units who possess decreased ability to adapt to darkness, in order to avoid the hazards incident to the injudicious employment of men whose night vision is below the average.

Aviation medicine

The study of the new field of aviation medicine reveals some new facts but mostly known facts widely extended. It is a specialty within itself in that it requires considerable knowledge of several specialties, some of them quite highly technical; for example, like ophthalmology and otolaryngology.

To quote from the Technical Manual,[12] "A defect which may be considered as minor or insignificant in the ordinary walks of life may be serious indeed for the pilot of aircraft." I might add, that we cannot be too careful in these examinations; a slip might cost the lives of all in the plane, the plane itself, and, it is quite possible, the lives of many in the army on the ground. To quote again:

> "In all probability there is no other type of physical examination where the time element is so important, where the adage 'make haste slowly' applies more forcibly. A complete ocular examination is at best a tedious procedure and requires an infinite amount of patience on the part of the examiner and the examinee, and, where 'short cuts' are employed or decisions made hastily, the accuracy of the finding is at best questionable. The eye examination should be objective as far as possible. Obviously the type of examination given for this purpose differs greatly from the examination given for clinical purposes. To the trained ophthalmologist, the eye examination is very simple, yet one has to use care that because of its extreme simplicity one does not become careless. This is a small matter, however, since relatively few trained ophthalmologists will be giving these examinations."

Fourteen steps are outlined on page 69 of the Technical Manual in the examination of candidates for flying training in the air service. It might be well to note them: Visual acuity, depth perception, Maddox rod with screen test at 6 meters (please note that it is the rod and screen test, otherwise serious errors will creep in), red-glass test, prism divergence, prism convergence and associated parallel movements, inspection, pupils, accommodation, angle of convergence (not PcB), central color vision, fields of vision, refraction, and ophthalmoscopic examination. It will be noted that this is quite a comprehensive examination for a physician who is not an ophthalmologist. Thus, special training is in order for the flight surgeons.

With the enormous number of flight surgeons required it would seem that perhaps the civilian medical schools and the eye hospitals could greatly assist the armed forces in this rapid education in the basic requirements in ophthalmology, which composes the most highly technical division in the subject of aviation medicine.

Color vision

In the matter of color testing and color vision, more instruction should be given in ophthalmologic training. A number of flying personnel are passed by the medical examiners who are "color unsafe." A number of cadets are admitted to West Point whose color vision is defective. This situation is exceedingly dangerous and

slows down the war effort. Likewise, it is casting an aspersion on the medical profession. There is some slight excuse for it in that, so far as I am aware, there is no clear-cut and decisive outline of a method to be used in testing color vision. There are no standardized nor accepted criteria upon which to work. The confusion in the terms used expressing color vision and deficiencies thereof is confounding. Nevertheless, the young ophthalmologist should be taught and required to know the subject well enough to make an efficient color-vision test for the services and to make a pròper diagnosis of the deficiencies expressed in proper terms. It would be a real help to the armed services if a pamphlet could be forthcoming expressing briefly, succinctly, and practically, the essentials that the doctor of medicine, making such tests, should know about color vision. Anyone who attempts to inform himself encounters theories as to how color perception is perceived, or different terms meaning the same thing and the same terms meaning different things. Those among you who have not experienced military service can scarcely appreciate what a boon it would be to the armed services, and hence to our country in time of need, if something that is practical and definite could be given to the doctors going into the Army and Navy. A quick and efficient method of testing color vision is a great need. Tests requiring extensive expenditure of time often mean hasty and inefficient examinations and faulty findings.

War ophthalmology

War ophthalmology differs but little from that of civilian life. The *facilities* for taking care of eye injuries, however, differ greatly. The utmost ingenuity must be exercised by the surgeon in overcoming the difficulties of the situation.

Vail[13] mentions in an editorial that at base hospitals a thoroughly trained ophthalmologist should be stationed with equipment necessary for extraction of foreign bodies from the eyes and all other care of the injured, sufficiently serious to be returned to the base. This is a problem which has not been answered as yet as to how the wounded that require hospital care can be gotten to the base. I have not the slightest doubt that the medical departments of the Army and Navy have formulated in minute detail the plans for such care. *But,* they are probably largely on paper. Having gone into a world war with probably almost total lack of anything approaching adequacy, good sense will tell us that the medical department of the Army has found it physically and humanly impossible to prepare properly against the vicissitudes of troops in the Arctic, on the Equator, and in the burning deserts, all requiring the greatest difference in the handling of such a colossal problem. How are men with seriously wounded eyes to be transported to a base that in order to be free from bombing has to be possibly a thousand miles away, or across seas, from the combat area? Here can be accomplished the plastic work for restoration of appearance and function, both of almost equal importance. The importance of adequate first aid in the combat zone to prevent infections, of rapid transportation and care en route of the wounded to the base, requires the almost perfect coördination of many departments of the army. With an army suddenly expanded from what was merely a national constabulary in *point of size* to one of many millions, the result has been a dreadful lack of officers with military experience, discipline, and training. It cannot be otherwise but that confusion, inefficiency, and slowed-down transportation will result in combat areas. It is inevitable that many of the wounded will be a long time reaching the base, and

that first aid is all they will get for some time. Such is the price our fighting men have always paid for unpreparedness.

The laboratories throughout the United States in the Army and Navy are originating and constantly working on relatively nonbreakable substitutes for glass. Much progress is being made. Plastics and resins are being utilized for lenses with some success. They are relativly unbreakable, which is a very great advantage, but they have other physical characteristics that are undesirable. For example, they are soft, and hence scratch easily; they do not retain their shape, and hence the dioptric strength does not remain constant, or the material becomes discolored by brilliant light and is less clear.

It is obvious that a substitute for glass for ophthalmic and other lenses for windshields and windows for airplanes, and so forth, which has not the above-mentioned disqualifications, will be of the utmost value.

Despite our criminally shortsighted preparation for war, which has heretofore been as inevitable as death, I think we are better prepared than in former wars, but relative to the remarkable preparation of our enemies, I doubt it. Vail invites attention to the suggestions of Sir Richard Cruise that 50 percent of the eye injuries could be prevented by some form of eye protection. The officers of the combat services say vision is impaired by such protector devices. The line officer is correct but that is only one of the reasons soldiers go into battle without many of the protections they might, could, or should have. These soldier lads have to carry it all on their backs. Only those of you who have been in battle can realize what that means, fatigue near to exhaustion, hunger, thirst, intense heat or cold, a weariness of soul and body which no civilian can understand, with a battle at the end of it. He throws everything away he has but his fighting tools, and no officers can stop him nor do they try. Sir Richard was correct but his viewpoint erroneous. Foot soldiers have always so stripped for battle; otherwise their fighting efficiency is much impaired and, of course, that cannot be.

Aviation ophthalmology

Major Weaver[14] in a personal communication states that high velocities, high altitudes, low oxygen tension, even when using an artificial oxygen supply (extreme cold, minus 68°F. at 35,000 feet), and reduced barometric pressure, together with night flying for combat observation and bombing, have created a number of new and basic problems, the most important one perhaps being fatigue. These problems have been studied for years by foreign powers, but in our own country not until very recently have laboratories and other facilities been available for our army to make the studies so urgently needed to solve these complicated and diverse matters. It is obvious that a group of men in many special branches of science are required to study these matters and the facilities of thoroughly equipped laboratories are necessary. In our country no adequate group of scientists or laboratories was available until about the time or shortly after the enemy had attacked and seriously crippled us. We were wasting billions of the people's money in political works administrations but only a trickle for that essential necessity for our national safety—scientific study of war and in preparation for war.

Many studies have been made since the recent facilities have been supplied but there are so very many which have not been made because of lack of time. We know research cannot be carried out hurriedly.

According to Weaver, "the factors peculiar to aviation which influence vision

may be listed as anoxia, decreased barometric pressure, temperature changes, acceleration, fatigue, nutrition, physical fitness, and certain drugs."

As to *anoxia,* it would seem that the direct effect upon the eye is closely related to the effect upon the central nervous system, the cerebrum in particular. Since the visual part of the eye is an integral part of the brain we may assume this to be true. Cerebral function is less effective and slower, depending upon the degree of anoxia, and so is vision. Visual acuity and color vision are lessened; perhaps the latter is obliterated. Night vision becomes less efficient. The visual field is reduced in size. Fatigue of the nervous system and, in consequence, of the whole body ensues.

Toxic states, resulting from infections, drugs such as the sulfa drugs, alcohol, and so forth, render the flyer less efficient. The toxins of fatigue, which occur naturally more promptly in those not in "hard" physical condition, also greatly contribute to early lessening of the efficiency of the flyer. No doubt the "poisons" of civilization must contribute to this condition; I refer to tobacco, tea, coffee, and alcohol in excess.

Is it not obvious that the best preventive of eye disabilities for the armed services and in particular the air forces of these services, is a strong body, a strong mind, and a pair of strong eyes for that strong body and mind to see with? Isn't it equally obvious that in order to have this, the physical training of these youths should begin at the beginning of life? When they enter school it should be continued and given equal value with mental training. If this had been done by the schools from the primary grades to the end of high school and by the colleges from their freshman year through the senior year, would we have so many lads disqualified for the air forces because of myopia and undeveloped bodies? I think not. Why did the schools neglect our youth? I do not know the answer. Do you?

Long-continued anoxia, even of low degree, leads to cortical changes, which though irreversible may be subclinical because of the great number of reserve cells in the cortex; similar changes may be inferred in the eyes, particularly in the ganglion cells. It is stated that the time required for irreversible changes in the cortical cells in acute anoxia is from 5 to 7 minutes.

As of this date, I am not aware of observations or experiments showing the effect of anoxia upon color vision. One must assume it is profound. According to Weaver, pilots have reported their color vision dulled as noted in signal lights after high-altitude flying. The lack of accurate knowledge concerning color vision prevents the acquiring of necessary information by observation and experimentation.

It is unfair to assume that those who have slight color-vision deficiencies may be dangerously low in color perception in the presence of anoxia, even of low degree. We are more aware of the effect upon the motor functions of the eyes—accommodation, the vergences, and the highly associated movements of the eyes—all of these may be disturbed. Minor motor anomalies may become serious major ones. I have observed a number of cases of exophoria due to divergence excess in those who have been flying for several years. They had been carefully examined before being given the responsibility and at intervals since. It has seemed to me that it has been acquired since they began regular flying and spending long hours at moderate airplane altitudes, 8 to 12 thousand feet. The majority of cases I have seen are not due to convergence insufficiency but to divergence excess. Could the anoxia, coupled with long-distance vision in which the divergence is presumably under a strain, have

a bearing on this? It is a fascinating hypothesis. We know, of course, that vertical phorias are less amenable to control than the lateral ones. Exaggeration of these vertical phorias, by anoxia, could disorganize binocular single vision very quickly and very effectually. Our knowledge of the effect of such disturbances upon convergent squint and less perhaps upon divergent squint will enable us to understand this easily.

Quoting from Weaver, who quotes Rickenbacker during the first World War:

"Another of those little precautions which might spell the difference between life and death, was the habit I forced on myself, always to make two complete circles of the landing field before landing at the end of a patrol from 15,000 feet."

He had observed that his landing ability was impaired; this is due to a number of causes, impairment of the depth perception being perhaps the most important. Weaver states,

"Vertical deviations are of the greatest importance in landing since, with such displacement, depth perception which results from fusion and consequent stereopsis is not only lost entirely but if the flyer lands according to the information furnished by the falsely projecting eye, he may land high or fly his ship into the ground. In the air, under combat conditions, similar misinformation under conditions of anoxia may lead to equal danger."

We remember that a latent deviation becomes manifest and exaggerated under anoxia and becomes a complete tropia with diplopia or monocular vision due to suppression of the vision of the deviating eye. Fatigue, profound emotional disturbances, the unusual ocular conditions of flying will all add to the motor weakness.

Anoxia definitely slows dark adaptation. Long-continued subclinical anoxia or the cumulative effects of anoxia have not been exhaustively studied. Cold does the same thing. It has been observed that in exposure to extreme and long-continued cold the effects upon the nervous system are very similar to those from anoxia and are probably due to anoxia from the slowing of the heart and, hence, of the blood stream. It is thought that this is the cause of the extreme somnolence that overcomes those approaching death from freezing. In anoxia it is probably, Weaver infers, that the synapse is altered in the retina and in the tracts from the visual cortex to the extravisual cortex.

The importance of night vision and combat aviation are stressed by Weaver. He emphasizes, on the one hand, the use of the macular retina and cone vision for daylight and color vision and, on the other hand, perpipheral retinal or rod vision. The rods serve a colorless task in daylight and register movement and large objects. At night when dark adaptation has taken place and illumination is by the stars, the rods are the more active. They locate the object of low light intensity. When, however, fixation is shifted to the macula, the image disappears because the cones have a much higher threshold for light than do the rods. "Rods are one thousand times more light sensitive than cones under conditions of dark adaptation," says Weaver.[14] "They do not distinguish color but only degrees of brightness." At present it appears that the complete serial values of cone and rod vision, under dark-adaptation conditions, are not so important in classifying the night vision of an individual as is the calculation of rod-threshold value after a time interval in the dark of 30 minutes. Obviously, in aviation the light threshold of peripheral vision is by far the most important factor.

Rods do not see colored light in their corresponding colors but only as white light of different degrees of brightness.

Consider three lights to be of equal intensity, blue, red, and white. Using "pho-

toptic vision"—that is, "day eyes"—the white or red will appear brightest and they will be recognized as different colors. "But let the individual see these lights from a great distance under conditions of dark adaptation, such as the pilot lights of an enemy plane, the blue light can easily be seen; the white light is 1/10 and the red light is 1/100 of the blue light's brightness." The red light will not be seen at all under such conditions. Obviously, as Major Weaver writes, the blue light is the most dangerous as a blackout light and red the least dangerous. This is a matter of great practical interest to ophthalmologists.

Night vision may be materially retarded by anoxia and a reduction or absence of vitamin A. It must be remarked here that giving vitamins to a normal person does not improve his night vision.

In *changes in barometric and temperature pressure* a great many new and difficult problems have arisen. Because of the cumbersome helmets, oxygen masks, and goggles, vision may be interfered with to a very marked degree. There are a myriad of mechanical failures of the apparatuses to supply oxygen and heat to the pilot in a temperature of minus 68°F., and low barometric pressure at 35,000 to 40,000 feet. All these make efficient seeing difficult and inefficient but can be corrected only by improving the mechanical arrangements necessary for sustaining life at such altitudes.

Acceleration (speed), particularly angular acceleration—that is, as encountered in the pullout of a dive—precipitates in some flyers the "gray out" which warns of the imminence of "black out" or unconsciousness. These phenomena are dependent upon habitus and physical fitness of the individual, the length of time the physical forces act; the number of G's (units of gravity). The latter is directly proportional to the velocity of the plane and inversely to the radius of the executed turn.

"It is not known whether the factors involved are cerebral ischemia with resultant cerebral anoxia alone or whether the differential between the pressure of the cerebral and retinal arteries is of importance" (Weaver[14]).

Fatigue

Of all the subjects discussed, none is more important to the pilots than efficiency of vision. The first and most important factor is the pilot himself; how good a man is he? How physically fit is he? Has he any inherent physical or neural weaknesses? We have discussed the most important contributing causes, anoxia, acceleration, and combat; also the mechanical appliances, goggles, masks, and so forth, all reducing efficiency. They fret and worry the pilot and this contributes enormously to fatigue. Goggles with a common glass for both eyes do not so reduce the nasal visual fields nor the binocular field of vision. In such a goggle, removable plastic lenses of different colors may be utilized. Plastic lenses, being fire resistant and non-shatterable, protect the eyes in a crash and also against fire, the latter being particularly important in the "flash" fires of this war. In various naval engagements the number of eyes badly burned from the "flash" fires (the blast from an exploding shell, a gun or bomb, or explosion of gasoline in a confined place) has been considerable. Tremendous heat is generated for a fraction of a second. Also many eyes are injured by swimming in oil-covered seas. This appears to cause a severe conjunctivitis and in some cases mild keratitis. So far as I am aware no permanent injury has resulted from this cause.

The use of heavily tinted glasses is really necessary under certain conditions of flying. Flying above the clouds produces

the most intense glare from below; the sun from above is a different sun from the one we see from the earth, much more powerful and intense. The combination of the two is very trying and very fatiguing even to the strong normal young eyes of pilots and requires protection.

Major Weaver[14] mentions an interesting fact in connection with drugs of the emphetamine type, stating that Axis pilots are believed to be well supplied with such drugs. It appears that the advantages outweigh the disadvantages. One may understand how the nervous system may be whipped up for a temporary exploit. In time of stress, it might be advantageous. It would appear that abuse of such drugs might occur and that their use should be attended with great care. Any drug stimulating the nervous system naturally accelerates vision and the whole visual apparatus temporarily.

It is unnecessary to consider before a gathering of ophthalmologists the minor injuries to the eyes. Their treatment and disposition are the same in civil life except certain modifications in the military service which will suggest themselves to the ophthalmic surgeon.

Burns from heat or caustic agents require care that often cannot be provided in combat and probably for some time afterward. It is useless to think of special solutions; if one has water from his canteen he is fortunate. The crudest possible arrangements may be contemplated and met with where the good sense of the surgeon and the skill in his hands will be about all that he has to work with. It may be that all we have with which to wash the eyes is water from a stream or a water-containing plant. Such plants are often encountered in the tropical jungle—the rattan vine, for example.

As to neutralization of chemical burns, this is practically impossible and in military life would probably be utterly so. Prompt removal of excess alkali or acid by copious injection, should fluid be available, is desirable.

My experience in the jungle fighting in Mindanao, Bataan, and other parts of the Far East (which is far no longer) enable me to say that if you have any medical supplies it will be due to your fighting spirit that you obtain them and to your physical vigor if you have them in combat. I mean that you, yourself, personally, may have to carry them on your own back into the fight. Hence, it is idle to talk of having for first aid this favorite prescription, or that favorite irrigant. At the field station, if one is at hand, more adequate first-aid treatment may be had.

Gas burns in military life, when they do occur, are sudden and in great numbers, and only the most casual first aid is possible as a rule. We are familiar with sodium-sulphate solution (0.4 percent) for use in tear gas burns. For mustard-gas burn, use copious irrigation as promptly as may be possible; if available, 1.5-percent solution of sodium bicarbonate, saline, or boric-acid solution. Five-percent sulfathiazol ointment is advocated in cases of severe burns. I have had no experience with this.

Lewisite is probably more destructive than mustard gas. I am not acquainted with its late or latent effects as compared with those of mustard. Copious irrigation and subsequent symptomatic treatment are all that can be given.

We should bear in mind that contusion of the globe may follow explosions, mines, shells, bombs, and so forth, without much obvious damage to the external tissues and be prepared to deal with the many complications and sequelae which arise as a result of such injuries.

It would be well if all, not technically informed, should thoroughly familiarize themselves with local anesthesia for ***all***

ophthalmic operations. General anesthesia may be impossible or difficult to have administered. There are no operations upon the eye or adnexa that cannot be performed without severe pain if the methods of securing anesthesia by infiltration or nerve block are adequately known and followed. This statement applies to civil life as well as to military life.

It is interesting to remember the War Manual Number 3, written by Colonel Greenwood, Colonel de Schweinitz, and Colonel Walter Parker, which would be perused with profit by any of the doctors going into military service.

927 Seventeenth Street.

References

[1] Military Surgical Manual of the National Research Council for Ophthalmology and Otolaryngology. Philadelphia, W. B. Saunders Co., 1942.

[2] Philips, T. J. Delayed mustard gas keratitis. Proc. Roy. Soc. Med., 1940, v. 33, p. 229 (Sect. Ophth. 5).

[3] Mann, Ida, and Pullinger, B. D. A study of mustard gas lesions of the eyes of rabbits and man. Proc. Roy. Soc. Med., 1942, v. 25, Jan., no. 3, pp. 229-244 (Sect. Ophth. pp. 1-16); Amer. Jour. Ophth., 1943, v. 26, Dec., pp. 1253-1277.

[4] Love, Col. A. G. (M.C.), U.S.A. (Ret.). Personal communication.

[5] Matthews, Maj. John L. (M.C.), U.S.A. Ophthalmic injuries of war. School of Aviation Medicine, Randolph Field, Tex. (Unpublished).

[6] Duthie, O. M. Discussion on ocular injuries resulting from the war. Trans. Ophth. Soc. United Kingdom, 1941, v. 61.

[7] Gifford, S. R. Injuries of the eye. (Military Surgical Manual of National Research Council for Ophthalmology and Otolaryngology. Philadelphia, W. B. Saunders Co., 1942, Chap. 5.

[8] Wakeley, C. P. G. War burns and their treatment. Practitioner, 1941, v. 146, Jan., p. 27.

[9] Wiener, Meyer. Personal communication.

[10] Lovelace, Maj. W. Randolph II (MC), U.S.A., Wright Field, Dayton, O. Personal communication.

[11] Military Surgical Manual, No. 11, Ophth. and Otol., Chap. 3, p. 24.

[12] Technical Manual, 8-300 TC 44. War Dept. Notes on eye, ear, nose, and throat in aviation medicine.

[13] Vail, Derrick. Editorial, Amer. Jour. Ophth., 1942, Feb., p. 218.

[14] Weaver, Maj. E. M. F. (M.C.), U.S.A., Randolph Field, Texas. The importance of vision in aviation. War Med., 1943, Sept.

EMILE JAVAL*

An appreciation

Burton Chance, M.D.
Philadelphia

From the list of subjects in which your committee suggested you might be interested, I have chosen the name of one for whom I have profound admiration. It is that of Emile Javal, known to us as an ophthalmologist, but, to his countrymen he was also a hygienist, a politician, an educationist, and a social reformer. By his death, our profession lost a very distinguished member who, at that time, was known throughout the world as one who had taken an important part in modern ophthalmology.

Javal was born in Paris, May 5, 1839, and, at his death in his native city, had reached his 67th year. Possessing an unusual and active mind, from his boyhood he was attracted by every problem, physical or biologic, that came his way. He had wished that he might enter medicine, but was trained as a mining engineer. He became interested in the case of his small sister who was cross-eyed and amblyopic (strabismus had been a distinctive feature of several members of his family, including his father). For her he conceived the idea that by means of a stereoscope, binocular vision might be developed. The results which she attained from her brother's perseverance during several years reached a degree of development sufficient to convince him of the value of his observation. Impressed by that result he decided to abandon engineering, and forthwith took up medicine. Having his sister's case in the background of his mind, he early began to give his special attention to diseases of the eye. For a period he was one of von Graefe's pupils.

With the outbreak of the Franco-Prussian War in 1870, he entered into the service as a military surgeon, and continued therein until the end of the war. He then returned to his chosen field of practice but, having espoused decidedly liberal ideas, he began to interest himself in political affairs. In 1875 he was elected to the General Council of the nation, representing a county district. Later, gaining a seat in the Chamber of Deputies, he became noted both in the Tribune and in committees. All this, however, without interrupting his professional work. M. Zola received much inspiration for certain of his works from Javal's interests and knowledge, and during the Dreyfus trial Javal went to Rennes and exerted himself on the side of the accused.

In the meantime he translated into French, Helmholtz's "Physiological optics"; continuing with energy and ingenuity the study and treatment of strabismus, the result of which he published in 1896, a "Manuel theorique et pratique du strabisme." It may be stated that through Javal chiefly have we learned that the faulty vision of the squinting eye frequently may be improved and that, by dint of compulsory education, through practice with stereoscopes and other educative exercises, the power of fixation may be acquired and true binocular vision established even in long-untried cases. Javal, accordingly, may be counted the patron saint of orthoptics. He warns us, nevertheless, that the time and trouble involved in achieving good results are out of all proportion, sometimes, to their value at the ultimate attainment.

* Read at the annual dinner of the New York Society for Clinical Ophthalmology, Tuesday, May 25, 1943.

In addition to these particular subjects, he was a persistent advocate of reforms in the hygiene of schools, especially toward the conservation of the sight of children.

My first knowledge of Javal was gained while inquiring about a well-worn instrument, called an "ophthalmometer," which I saw at Wills Hospital while still an undergraduate. It is such an instrument with which Javal's name is more definitely connected than with his extensive work on strabismus. The principle of the ophthalmometer consists in affording means for the measurement of the curvature of the cornea and the lens (a keratometer it really is) by observing the images of special parts reflected from their surfaces. The estimation of such images was not an original discovery by Javal, for, years previously, Young and others had devised instruments for the purpose, and Helmholtz more recently had perfected an instrument which Donders deemed to be "one of the greatest treasures for which we are indebted to his genius." Yet, today, we owe to Javal and Schiötz, his Norwegian collaborator in the laboratory of the Sorbonne, the simplified instrument with which we can rapidly study the cornea's curvature.

Their first perfected instrument was exhibited at the International Medical Congress, held in London, 1881. The ophthalmometer took a place among the approximate tests in the practical working out of ametropia. Its indications are definite and the ease and rapidity with which these may be obtained betokens its usefulness. The results which can be reached with scientific precision and accuracy, in the case of astigmatism in aphakic eyes, assure us of its distinct value. It has positive limitations in usefulness, however, for its does not give the refractive condition as a whole, nor furnish indication as to the existence of myopia or hyperopia; nevertheless, while giving with exactness the radii, in all meridians, of curvature of the cornea, it manifests the directions of the principal ones.

The history of the Javal ophthalmometer may be found in a volume, edited by Javal, entitled "Mémoires d'ophtalmometrie," containing a number of papers by many writers, European and American, including Theobald and Swan Burnett, who became ardent advocates of the use of the instrument.

We are indebted to Javal for the adoption of the metric system in the dioptric numeration of lenses, and the abandoning of the old inch measures. He suggested also gilded rims for the concave lenses and silver for the convex. He devised the star or fan for the estimation of astigmatism. He urged the increased utilization of cylindric lenses even in low degrees of error.

In 1885, at the end of an election campaign, by a sad irony of fate, in spite of skillful treatment, his right eye was blinded by glaucoma. In the succeeding years he suffered many transitory attacks in his left eye. In 1897, the excitement of the Dreyfus trial at Rennes precipitated the final attack. He was kept more or less under the influence of pilocarpine during 15 years of active work. In 1900 he went to England for treatment, under the direction of Priestley Smith. An iridectomy gave only temporary relief, and, during the succeeding five months, in spite of all efforts, the eye became blind. On his return to Paris, by his own desire, the cervical sympathetic was divided, but with no benefit to his sight.

This terrible affliction did not dampen his spirit. On the contrary, with philosophic fortitude he promptly set himself to study the means by which the lot of those who lost their sight during active adult years might be ameliorated. Javal early found, as did Helen Keller, that

"the greatest curse of the blind is not blindness, but idleness." After three years of experience he issued a little book which he called "Entre aveugles." It has been translated into many languages; the English version bears the title "The blind man's world"; my American edition, published in 1905, "On becoming blind." The author counsels the blind and their friends on such matters as "Replacing sight by other senses"; "Household and professional occupations"; "Marriage"; "Neatness, hygiene, health"; "Dwelling"; "Meals"; "Watches and clocks"; "Walking"; "Tandem tricycle"; "Travelling"; "Reading aloud"; "Handwriting and typewriting, including raised, or embossed Braille"; "Music"; "Games"; "Tobacco"; "Esperanto"; "The sixth sense"; and a chapter on the "Psychology of the blind."

The book is a fine instance of valuable work planned and carried out under a calamity great enough to overwhelm most individuals. In reading between the lines one is impressed by the sincerity of the afflicted gentle soul whose efforts to help others are but expressions of his determination to conquer his own helplessness. Friends returning from visits to France repeatedly told me of their having seen Dr. Javal on his tricycle wheeling through the streets of Paris; and, medical men of their conferences with him.

In 1894 Javal edited and prefaced an edition of the ophthalmologic works of Thomas Young which Tscherning had translated into French, published in Copenhagen.

During these latter years Javal was preparing another important work "Physiologie de la lecture et de l'ecriture" which appeared in 1906. It describes the evolution of written and printed characters, including those of stenography, musical notation, and relief-writing for the blind. He discussed the principles on which letters should be formed to afford the maximum legibility and he indicates how handwriting might be so faulty as to become "an offence to common sense." The first edition was exhausted in three months.

These works show how keen and how purposeful was Javal's interest in many things; "Esperanto," for instance, and "Spelling reform," and, always unceasingly, means for improving the condition of the blind.

Javal was a deeply religious man, maintaining the highest ideals in his daily life. Many honors were granted him; honors that indicate the appreciation of the world for his service to medicine and to ophthalmology. In 1885 he was elected to the "Acadamie de Medecine," and honorary member of the Opthalmological Society of the United Kingdom of Great Britain, having been invited to deliver the Bowman Lecture in 1907. His health was such, however, that he could not undertake the journey. Death soon followed from the effects of gastric cancer, January 20, 1907.

My desire to tell of the life and works of Emile Javal rests on a long-held belief that he is preëminently the exemplar for the new-education of the sightless and the guide leading the blind onward in self-dependence. It would be natural for him, and for ourselves to expect him, to offer advice to practicing ophthalmologists, something of the means with which to lighten the misery of their unfortunate patients and others. The little book is packed with suggestions for ways to enable the adult blind to "live with the least possible evil with their infirmity." Children, as we all know, are reported on early, and are sent to schools for their special instructions, and are taught all the arts and maneuvers enabling them to enter life with remarkable independence.

Javal's avowed purpose was to write for the family of the blind, offering to

them helps attainable at the smallest cost, with which they themselves can lighten their burden in living with the blind one. The chief burden of the blind, as he knew and as we, too, know, is their forced dependence on others.

He begs us "to resist" what he calls "the barbaric tendency" to leave our patients with the hope of recovery, whose course toward blindness is certain, that by injections of one or more kinds of drugs, electrical currents, or useless internal medicine, their condition can be relieved. To give consolation by a placebo treatment to an incurable is to prevent him from arranging his life in anticipation of the fatal outcome. It is more humane to prepare them, little by little, for their fate. He, accordingly, wishing that there might be done for others what he would have liked to have had done for himself, sent forth this little book with the hope that it might serve to render easier some ill-fortune like his own and thereby give to himself the special consolation of having helped others.

The appliances and other helps which he recommended, and all that have been devised since his time, can be obtained through the Blind Societies in any city in the country. No person is too old to learn to use them; the struggles of the teachers are concerned almost entirely in enforcing the will of the pupils to accept instruction and to carry on after the lessons have been accomplished.

Out of my great admiration for Javal, my desire has been to make a special appeal to ophthalmologists to consider the future of those whose sight is irrevocably falling into helpless blindness. Just here there occurs to me the knowledge that two ladies, well beyond 60 years of age, suffering from high-grade myopia—one with well-preserved backgrounds—but who has had repeated extravasations—the other with ragged backgrounds and cataractous lenses, each has in the past two years become so skillful in typewriting as to be able to carry on correspondence with their friends and conduct their daily business notes. A gentleman, when forced to reside at a great distance from civilization, took up bee-keeping. Later, when I extracted his lenses, so brilliant became his sight that he, at times, regretted that he had had them extracted—so simple and easy had life become, he was no longer his own hero.

For several years I have been one of the Pennsylvania Home Teaching Society for the Blind and have the honor to be the President of the Board which manages the affairs arranged solely for the benefit of the hundreds of adult pupils to whom annually thousands of visits are made by a staff of competent, though entirely blind teachers.

My allusion to these facts arises from the likelihood, and indeed the probability, of a great increase in the number of the blind following the present worldwide cataclysm—both civilian, and through "grim-visaged War."

In the Archives of Ophthalmology for April, 1939, in commemoration of the centenary of Javal's birth, James Lebensohn published a sympathetic tribute to the master. In that you will find what I have wished I might have had the time to tell of his personality, to add to the brief, matter-of-fact account I have given.

317 South Fifteenth Street.

ENDOPHTHALMITIS PHACO-ANAPHYLACTICA*

RICHARD G. SCOBEE, M.D., AND HOWARD C. SLAUGHTER, M.D.
Saint Louis

It was in 1934 that Burky[1] first reported his ingenious method for the production of experimental endophthalmitis phaco-anaphylactica in rabbits. In his report he also gave a brief but complete summary of the work and ideas on the subject up to that time. There is no need for its repetition here. To the best of our knowledge, his work has not been confirmed in the literature, and hence this paper in confirmation.

Burky[2] had previously noted that rabbits given intracutaneous injections of staphylococcus toxin developed not only an antitoxin for the toxin but also a hypersensitive state to the beef broth in which the toxin was produced. He reasoned that if such a poor antigen as beef broth could be made active by the intermediary action of staphylococcus toxin, then this same toxin might have a similar action on lens substance.

The experiments which he then devised bore out his idea, the results confirming the hypothesis of Verhoeff and Lemoine[3] concerning the allergic nature of endophthalmitis phaco-anaphylactica. He demonstrated that such an allergic state could be attained through the intermediary action of bacterial toxin.

It is not necessary to describe his technique here in detail; those interested should read his original paper.[1] It is our purpose merely to confirm Burky's work, the confirmation being the first step in a series of experiments having to do with the possible relation between endophthalmitis phaco-anaphylactica and sympathetic ophthalmia. DeVeer[4] (1940) wrote an excellent article on this subject, speculating upon a possible relationship between the two entities and citing several case histories in support of his speculations; no experimental work was done, however.

EXPERIMENTAL

According to the method of Burky, both a lens extract (of beef eyes) and a lens-broth toxin were prepared, an HA strain of Staphylococcus being used for the latter. Seven rabbits were then prepared for intracutaneous injection by depilation with barium sulfide. Two rabbits were given 0.5-c.c. intravenous injections of the lens extract each week. The others received at weekly intervals 0.1 c.c. of lens-broth toxin in one cutaneous area and 0.1 c.c. of lens extract in another cutaneous area. Reactions were recorded by measuring the maximum diameter of erythema, swelling, and necrosis in millimeters.

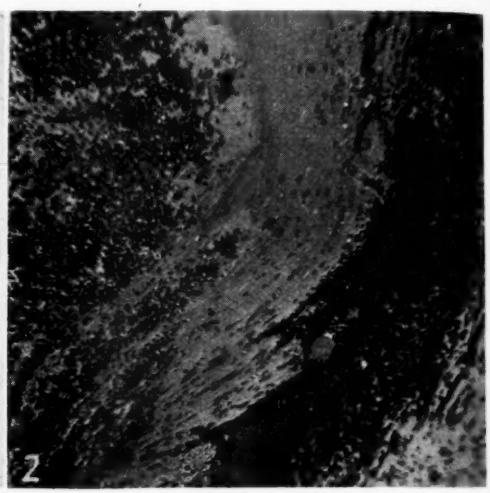
The first injections were followed by erythema and swelling, which appeared in from 24 to 48 hours. It is interesting to note that, in contrast to the observations of Burky, the cutaneous reactions of our group of rabbits seemed to decrease rather than increase with each subsequent injection, yet the final outcome was the same; that is, a clinical and histologic picture of endophthalmitis phaco-anaphylactica. Also in our series, necrosis was absent or minimal. These findings were the same both for the lens extract and the lens-broth toxin. Burky found that the majority of his rabbits attained their maximum sensitivity (cutaneous) following the fourth injection. Because of the lack of any marked skin reaction indicative of a developing cutaneous sensitivity, our

* From the Washington University School of Medicine, Department of Ophthalmology, Oscar Johnson Institute.

series was carried to seven injections in an unsuccessful attempt to demonstrate this same cutaneous sensitivity. A small erythematous area measuring from 0.5 to 1.0 cm. would occasionally mark an injection site 24 or 48 hours later. There were numerous occasions on which not even this small skin reaction could be noted.

and remained there when the syringe method was used; another factor was, perhaps, that of internal swelling of the lens material produced by the injected aqueous, forcing apart the lamellae.

The eyes were enucleated at varying times following this experimental trauma. Two eyes had to be removed on the fourth day to prevent their rupture, and one



Figs. 1 and 2 (Scobee and Slaughter). Endophthalmitis phaco-anaphylactica. Fig. 1, Low-power section of entire globe showing reaction of endophthalmitis phaco-anaphylactica. Fig. 2, Higher-power view of section of figure 1 as indicated by lines. The lens substance shows monocytic invasion, and dissolution can be seen at the left. The heavy, dark-staining infiltrate at the right is the surface of the ciliary body. A zone of relatively clear cortex is between the two infiltrative zones.

At the completion of this series of sensitizing injections, the right eye of each of the rabbits was needled with a Ziegler knife-needle, a crucial incision being made in the lens. Two days later, the left eyes were needled according to Burky's method of aspirating aqueous in a hypodermic syringe and then injecting the aqueous thus obtained deep into the center of the crystalline lens. It was noted that although similar clinical and histologic pictures developed in both eyes of each sensitized rabbit, the reaction was definitely more marked in the left eyes (traumatized by Burky's method). This might be expected, since aqueous was brought into contact with lens material

ruptured during its removal. The same thing happened in Burky's series. The remaining eyes were enucleated on the tenth day, when clinically all showed an endophthalmitic reaction of varying degrees of severity. There was suspicion of secondary infection on clinical grounds in two of the eyes, and this was confirmed histologically by the predominantly polymorphonuclear character of the infiltrate in them. Eyes from the control rabbit showed only a normal response to the trauma of needling. Rabbits 9 and 11 were those which received the intravenous injections of lens extract and they showed practically no reaction other than that due to the trauma of the needling.

Dr. T. E. Sanders was kind enough to study the group of eyes pathologically, and his report was as follows:

"The reactions in these eyes varied from very mild to extremely severe. In all cases, the reaction was chiefly localized in the anterior segment. In the mild cases, there was no particular inflammatory reaction. There was a wound of the anterior lens capsule with varying degrees of degenerative cataractous formation. This process usually extended around to the posterior capsule. There were varying degrees of attempts at healing in the anterior capsular wounds by a fibroblastic reaction. Inside the posterior capsule, a peculiar cellular ingrowth formed almost a posterior epithelial layer; this apparently arose from the region of the equator. The posterior subcapsular cells were present in the eyes of rabbits receiving the intravenous injections as well as those which had the intracutaneous ones. There was a homogeneous eosinophilic substance in the anterior chamber.

"The eyes showed a chronic inflammatory reaction, chiefly in the area of the lens trauma. This consisted of a heavy infiltration of chronic inflammatory cells in the area of liquefied lens and extended out somewhat into the more intact lens substance. There were relatively dense posterior synechiae but relatively little infiltration into the iris tissue in the milder cases. The infiltrate was predominantly monocytic, with a few lymphocytes and polymorphonuclear cells. There was no evidence of necrosis; varying degrees of vasodilation of the iris vessels were noted. A moderate number of monocytes were seen on the endothelium of the cornea, on the surface of the ciliary body, and on the nerve head. This distribution was essentially that of chronic endophthalmitis.

"In the more severely involved eyes, a marked keratitis was present near the

TABLE 1
SUMMARY OF OCULAR REACTIONS

Rabbit No.	Injections before Needling	Cutaneous Sensitivity to Lens Extract at Needling	Ocular Reactions		Day of Enucleation
			Clinical	Histological	
Group I. Lens-broth toxin and lens extract intracutaneous injections					
1	7	Waning	Positive	Positive	10th
2	7	Positive	Positive	Positive	10th
3	7	Positive	Doubtful	Doubtful	10th
5	7	Waning	Positive	Positive O.S. Secondary infect. in O.D.	10th
6	7	Waning	Positive	Positive	4th
7	7	Positive	Positive	Positive O.D. Secondary infect. in O.S.	10th
8	7	Waning	Positive	Positive	4th
Group II. Lens extract injected intravenously					
9	7	Negative	Trauma	Negative	10th
11	7	Negative	Trauma	Negative	10th
Group III. Normal control rabbit					
10	None	None	Trauma	Negative O.S. Secondary infect. in O.D.	10th

limbus which included all the layers of the cornea and seemed to have no relation to the site of corneal penetration from the needling. On one side, this keratitic area was so thin that early ciliary staphylomata were seen. Over the surface of the retina in the more severely inflamed eyes could be seen monocytic cells. In the secondarily infected eyes, the polymorphonuclear cell was quite prominent in the infiltrate.

"In the group of eyes not secondarily infected, the pathologic picture was one of endophthalmitis, with predominantly a monocytic infiltration, hence a 'chronic endophthalmitis.' "

Comment

Friedenwald[5] has stated that the character of the cellular infiltrate is of some value in distinguishing the findings in the allergic animals from those in accidentally infected control animals. In the latter, the cells are predominantly polymorphonuclear, whereas in the allergic animals, they are predominantly monocytic.

Summary

Herewith is presented confirmation of Burky's work of 1934; namely, that cutaneous sensitivity to lens substance in rabbits can be induced by repeated and coincident injections of staphylococcus toxin and lens substance. When the eyes of such sensitive rabbits are needled, a clinical and histologic picture develops that resembles endophthalmitis phaco-anaphylactica in man. Our series did not show the cutaneous sensitivity which Burky mentioned, yet end results were the same. We report this confirmation of Burky's work as the first step in a series of experiments having to do with endophthalmitis phaco-anaphylactica and sympathetic ophthalmia.

640 South Kingshighway.

References

[1] Burky, E. L. Experimental endophthalmitis phaco-anaphylactica in rabbits. Arch. of Ophth., 1934, v. 12, Oct., no. 4, p. 536.

[2] ———. Antitoxin content of rabbit serums immune to staphylococcus toxin and the precipitin reactions of such serums. Jour. Immunol., 1933, v. 25, p. 419.

[3] Verhoeff, F. H., and Lemoine, A. N. Endophthalmitis phaco-anaphylactica. Trans. Internat. Cong. Ophth., 1922, p. 234.

[4] deVeer, J. A. Endophthalmitis phaco-anaphylactica and its relation to sympathetic ophthalmia. Arch. of Ophth., 1940, v. 23, Feb., p. 237.

[5] Friedenwald, Jonas. Cited by Burky.[1]

VISUAL ACUITY AT LOW BRIGHTNESS-LEVELS*

MATTHEW LUCKIESH, D.Sc., AND A. H. TAYLOR
Cleveland

Visual acuity, or the ability to distinguish fine detail, is often of little importance at very low brightness-levels. Contrast sensitivity, or the ability to distinguish small differences in brightness, is commonly of great importance at low brightness-levels such as are encountered outdoors at night. For example, visual acuity has been greatly overemphasized as compared to contrast sensitivity in the testing of automobile drivers for seeing at night. Also there are many problems of seeing in war activities which involve concealment, dark adaptation, and very low brightness-levels at night under moonlight, starlight, and even dark, overcast skies. Lookouts on ships, pilots and observers in airplanes, and drivers of trucks might well be tested for contrast sensitivity. Researches by the authors have revealed a wide diversity in this function even among subjects who have the same visual-acuity or visual-efficiency ratings by the usual methods.[1]

On the other hand, there are some activities in which it is necessary to distinguish fine details under the least amount of light, or lowest brightness, that is practicable. Scales of instruments, maps, charts, and other details must be read with accuracy and as easily as possible under blackout or dimout conditions. In various researches[2] in connection with problems of seeing in wartime, the authors have had to study the effects of illuminants of various spectral characters upon visual acuity. Subjects vary greatly in their ability to see at low brightness-levels, but the data presented herewith were obtained by subjects who appear to be fairly representative. However, the quantitative results are less important than the decisive differences among illuminants. For this reason the results are presented in diagrams rather than in tables.

In figure 1 are shown typical results obtained with the A.M.A. test chart for seven different illuminants representing a wide range in spectral character. Under each illuminant the subjects were adapted for 30 minutes at approximately the brightness-level at which the largest letters could be distinguished at the test distance of 20 feet. The chart was located in the center of a circular, white field which subtended an angle of 20 degrees. This surrounding field and the test chart were illuminated by the same illuminant. After determining the test line which could barely be read, the brightness of the white background of the test chart was determined by someone other than the subject. Then the background brightness was increased and after adaptation for a standard length of time, about five minutes, the subjects again determined the test line which could barely be read. This procedure was continued for all the lines of the test chart. In all cases foveal vision was used, although in some cases apparently a slightly high visual acuity was obtainable by slightly averted vision.

On figure 1 the illuminants designated by the letter *F* are regular fluorescent lamps. The tungsten-filament lamp was a regular 150-watt lamp and the photographic red lamp was a 40-watt tungsten filament in a red-glass bulb.

It is obvious that as the illuminants progress in integral color from blue to red, there is a progressive decrease in the

* From Lighting Research Laboratory, General Electric Company.

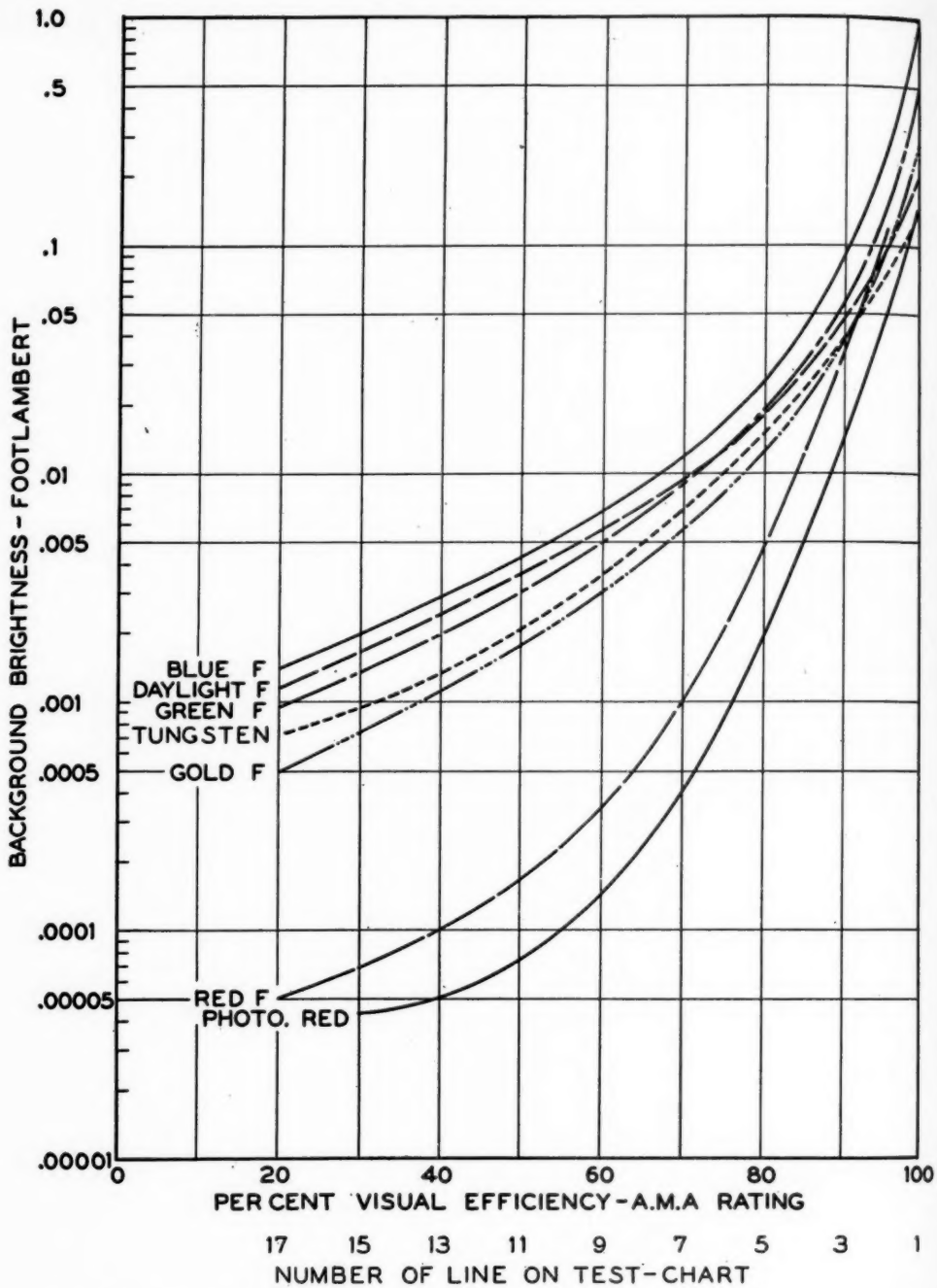


Fig. 1 (Luckiesh and Taylor). Minimum brightnesses obtained with different colored fluorescent (F) lamps, a regular 150-watt tungsten-filament lamp, and a photographic red lamp, at which various lines of letters of the A.M.A. test chart can barely be read.

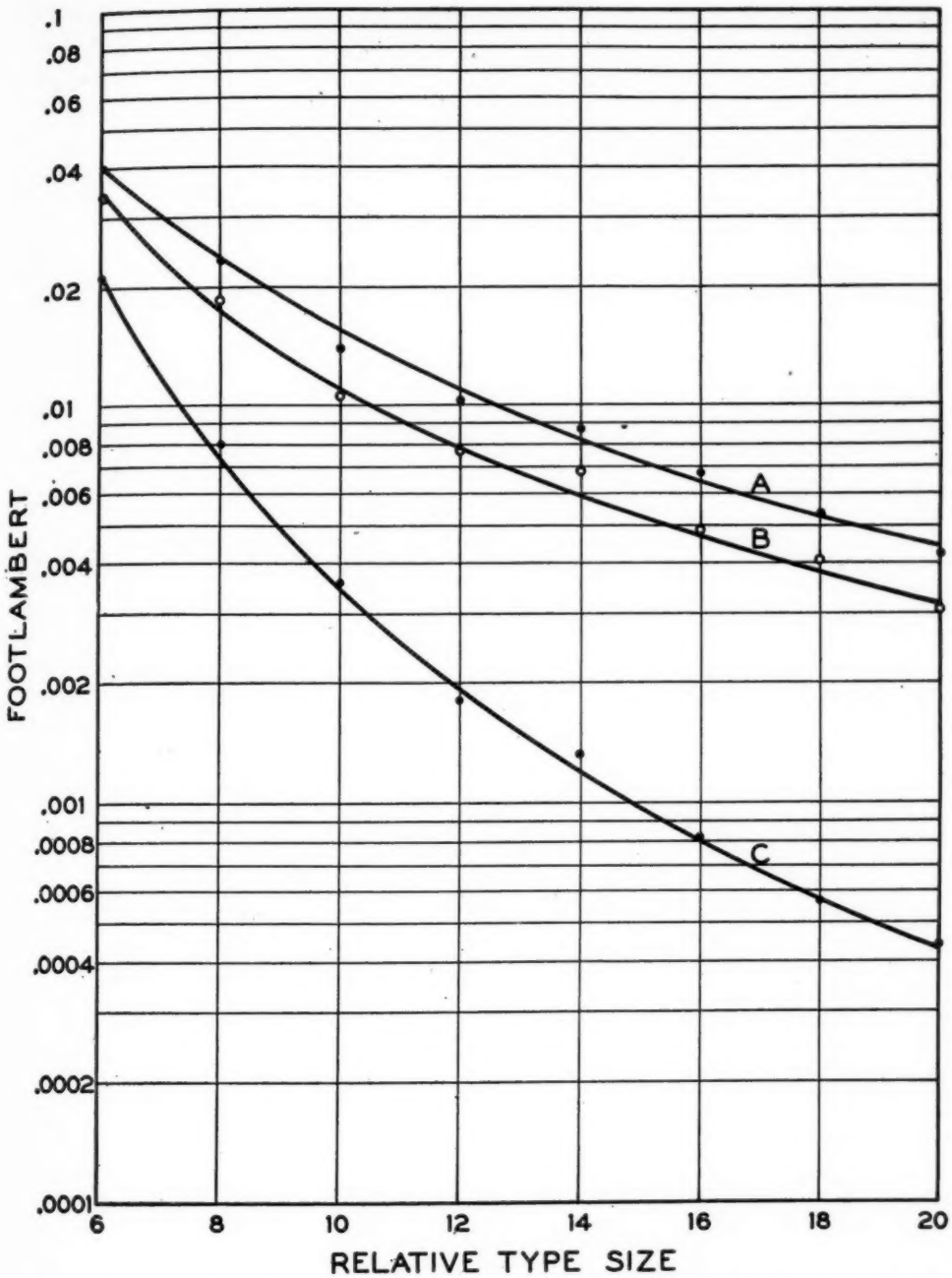


Fig. 2 (Luckiesh and Taylor). Background brightness required for slightly above threshold "readability" of type sizes with (A) tungsten-filament lamp at 105 volts (color-temperature approximately 3,050 degrees K), (B) the same lamp at 40 volts, and (C) the same lamp at 105 volts with Corning HR Signal Red no. 243 filter.

minimum brightness of the background of the test chart at which a given test line can be read. In other words, red light is more effective for a given brightness than the other illuminants. This is a very important matter in many wartime activities where it is necessary to see details with a minimum of brightness.

These data afford a basis for many recommendations. For example, suppose letters, numbers, or other details subtend a visual angle of 20 minutes, corresponding to line 9 on the A.M.A. test chart. The minimum background brightness for such details of maximal contrast (black on a white background) should be about 0.0035 footlambert for tungsten-filament light and about 0.00014 footlambert for red light from a photographic red lamp. This is an enormous difference, for the former brightness is 25 times the latter. Such great differences are of much practical value where concealment, visibility, and the maintaining of a high state of dark adaptation are involved. Of course, to insure certainty of seeing, somewhat higher brightness values should be used.

For those used to thinking in terms of foot-candles or in measuring foot-candles, a definition of the foot-lambert may be helpful. If a perfectly diffusing white surface had a reflection-factor of 100 percent, a foot-candle of illumination produces a foot-lambert of brightness. The white surfaces used reflected about 83 percent of the incident light and therefore had a brightness of 0.83 foot-lambert for each foot-candle of illumination. Therefore, if the foot-lambert values are multiplied by a factor of 1.2, they are converted into approximate foot-candle values.

In figure 2 are shown the results obtained for three illuminants for reading matter printed with black ink on white paper. The type sizes ranged approximately from 6-point to 20-point. Curve A is a plot of results obtained for a 115-volt tungsten-filament lamp operated at 105 volts, the color-temperature of the light being 3,050 degrees K. Curve B is for the same lamp operating at 40 volts. Curve C is for a fairly saturated red light.

In figure 2 are shown the background brightnesses for the three illuminants which are respectively necessary in order to read with certainty type of a given size when printed with black ink on white paper. Here again the great advantage of red light is revealed; that is, when details are to be distinguished with a minimum brightness.

In obtaining the data plotted in figure 2, the subject was seated at a table and the reading matter was inclined at approximately 45 degrees. The light was confined to the reading matter by means of a projector and iris diaphragm. The subjects were adapted for 30 minutes to the brightness of the reading matter, the brightness being just sufficient so that the printed matter was just below the threshold of "readability" of the largest type. After adaptation the brightness necessary for reading without uncertainty was determined. Then the next smaller type was placed in position. After a standard rest period the procedure was repeated.

The work of the authors has shown in other ways[2] the great advantage of red light in a variety of problems of seeing in wartime—blackouts, dimouts, instrument dials, charts, orders—where concealment is vital[3] and where dark adaptation must be maintained. The results have many other applications in indicating the visual size of details necessary if they are to be distinguished under very low brightness-levels.

Nela Park.

REFERENCES

[1] Luckiesh, Matthew, and Moss, Frank. The ability to see low contrasts at night. Jour. Aeronaut. Sci., 1942, v. 9, p. 261.

———. Contrast sensitivity as a criterion of visual efficiency at low brightness-levels. Amer. Jour. Ophth., 1939, v. 22, p. 274.

[2] Luckiesh, Matthew, and Taylor, A. H. A summary of researches in seeing at low brightness-levels. Illum. Eng., 1943, v. 38, p. 189.

[3] Luckiesh, Matthew. Blue light undesirable for blackouts. Illum. Eng., 1942, v. 37, p. 113.

ORTHOPTICS FOR THE INFANT SQUINTER, ONE TO FOUR YEARS OLD*

EDITH V. ROTH
Milwaukee, Wisconsin

I have been asked to take up the various aspects of our present orthoptic treatment of the infant squinter at the Milwaukee Children's Hospital. However, orthoptic treatment cannot possibly be divorced from ophthalmologic treatment, especially in the infant. Therefore, I shall discuss the ophthalmologic treatment of strabismus in children under four. I only hope that this will not seem too presumptuous on the part of an orthoptic technician. Any opinions expressed on points other than those applying strictly to orthoptics are, of course, the opinions of the ophthalmologists on the Hospital staff.

At the Milwaukee Children's Hospital, an immediate examination, even at six months of age, is considered advisable for several reasons: (1) There are the occasional cases of +8 diopters or more of hyperopia. (2) There are the many cases of pseudostrabismus due to a wide epicanthus, in which the ophthalmologist can assure the parents that, although there is a definite illusion of strabismus, none is actually present. (3) There are the cases in which a true strabismus is found, due to some organic anomaly or disease.

In all cases of squint that show high refractive errors under a 0.5 percent atropine cycloplegia, glasses are routinely prescribed. In this regard, it is always wise to keep in mind that even a baby will wear glasses if he truly needs them. However, nothing in the world will make a baby keep them on if he does not need them.

When the refraction has been done, the ophthalmologist generally turns the infant squinter over to the orthoptic technician with the familiar words, "Would you like to *try* to get a vision on this child?" With the average child of two to four years of age, a vision usually can be obtained, if not by one method or on that day, by another method, perhaps, or on another day. The illiterate E or "piggy" game as we call it, the Kindergarten chart, the Berens chart, the Landolt broken-ring test, or graded blocks can be used. I am told that the Baily visual-acuity test is often helpful with a child who has difficulty in understanding what is expected on some of the other tests. In this test, the child is asked to pick from a tray at his side miniature models which match the object-pictures on the chart. Of course, making a game out of any method

---

*From the Milwaukee Children's Hospital. Read at the second annual meeting of the American Association of Orthoptic Technicians, in Chicago, October 12, 1942.

will get results much quicker than expecting the child to sit up and pay attention to a chart on the wall, which many times will probably look like a lot of Greek hieroglyphics. In any child of this age, obtaining a satisfactory visual finding is a challenge and, as it constitutes the first introduction to the little patient, it requires all the resourcefulness at one's command.

When this has been done, the little squinter is generally turned over to the orthoptic technician for a diagnostic series of five or six visits. In the event that a visual acuity has not been obtained, the fixating eye, in a case of monocular squint, or the preferred eye, in an alternating squint, is always easily determined, except in a few cases of alternating squint in which there is no marked preference of either eye. At this time, the parent is instructed as to the form of constant occlusion to be used following the diagnostic series, if it is known that occlusion is indicated. In very simple language the parent is told that occlusion must be used in the better or fixating eye in order to prevent an amblyopia ex anopsia from developing or to improve the vision in an eye already amblyopic. When occlusion is later begun, we usually, in the beginning, use complete occlusion—a patch over the eye if no glasses are being worn or a Stark occluder attached to the glasses if the latter have been found necessary. Both are cut out of waterproof adhesive tape and shaped to the individual face or pair of glasses. When the vision is found to be 20/70 or better, a less complete, but still constant form of occlusion may be substituted by pasting an opaque paper or gummed, translucent mending tape to the lens of the better eye. The possibility of the child's peeking then must be carefully watched. If the complete occlusion on the face of the child who needs no glasses is found too harsh a treatment to be continued indefinitely, a pair of plano lenses may be prescribed for the duration. Of course, there is always a question as to whether or not the child will keep these on. Other forms of occlusion can then be easily devised by the ingenious parent, such as a soft wide bandeau or a net bonnet, with the patch attached.

Not only is this monocular occlusion considered important for the prevention or improvement of amblyopia but also for the prevention of secondary contractures of muscles, by giving the patient a sufficient amount of monocular exercise. In the case of alternating squinters, an alternate occlusion of first one eye and then the other is usually suggested in order to prevent the development of an abnormal retinal correspondence. When binocular vision is found to be possible, a fogging of the lens of the stronger eye is sometimes suggested. This fogging of the lens may be accomplished in numerous ways, such as by the use of Scotch cellulose tape, a stippling of colorless nail polish, a clip-on smoked glass, or a red kodaloid filter. All of these forms of partial occlusion are of course advised only for infant squinters who have a normal retinal correspondence of long standing.

When the vision, fixation, and the necessity of occlusion have been determined, at least an estimate of the angle of squint is made. At three to four years of age this is usually possible for distance and near vision with the use of a cover test; then, at the same time, monocular and binocular rotations are generally attempted. With a baby under three it is possible only to estimate the angle approximately by the Hirschberg method and to determine a marked paresis. To be sure, any testing attempted in the beginning must be in the nature of a game and in the spirit of play.

On the second visit, a near point of convergence with a colored pinhead or a light is easily elicited, and monocular and binocular rotations again attempted to

ascertain any limitation of motion or overshooting in any direction of gaze. The angle of deviation in the primary position for distance and near is again checked.

The next step is to evaluate the amount of binocular vision the child has in his everyday life. Here we are forced to digress from the routine testing and find out, before going further, if the child knows colors and can count. Needless to say, the Worth dot test, which is the simplest method of estimating the patient's binocular vision, is possible only on a child of three or older. But since it is basically a subjective test, we must determine how much we are going to be able to rely on the child's answers before attempting any actual testing. There are many children who will recognize red and possibly green in a familiar article and yet not be able to recognize it in the Worth four-dot test. We find it helpful to practice naming the colors with colored blocks, marbles, crayons, or even gum candies, before showing any colored lights, as we then know immediately if the colors can be recognized and named correctly. We also find it helpful to practice counting before doing any actual testing. To be sure, this all takes valuable time, but it is always time saved in the end.

On the second or third visit we usually attempt to obtain the measurement of squint and the kind of retinal correspondence on the major amblyoscope. Needless to say, this is not always possible in the case of a child under three years. With such fascinating targets as the lion and the cage, the butterfly and the net, the spider and web, or the bird and cage, we let the child show us where he puts them together, and then quickly flash to determine if the simultaneous macular perception is at his objective angle. If we stimulate the child, in the spirit of fun, to show us how good he is, we can usually determine, in repeated attempts, if he is actually telling us what he sees or merely attempting to please. In the beginning, we generally find the child eager enough, and yet not too much aware of what he is supposed to see, to obtain the correct answers without much checking on our part. Of course, our flashing to determine the type of correspondence must be quick, as the child's concentration at this age is always poor, despite his eagerness and interest. No findings can be relied upon at this time unless they are corroborated on repeated attempts.

All of our tests are repeated on successive visits until we feel that we are able to give an accurate report of our findings of the visual acuity, fixation, degree of angle, type of correspondence, and measurement of fusion and stereopsis. Even the latter measurement is quite possible if the child's correspondence is normal and his fusion is good. In this event, it is possible that some fusion training at periodic visits may be prescribed to stabilize the present fusion and perhaps acquire some amplitude. However, this is rarely the case, for the average infant squinter at the Milwaukee Children's Hospital is usually advised, after the diagnostic series, to return only for bimonthly check-up visits until he has reached an age at which he can accept concentrated orthoptic treatments. Any actual training given at this time is mainly to help the child to become accustomed to the clinic and the technicians and to determine the type of occlusion, if the latter has been found necessary. On these visits, when the vision, fixation, angle of deviation, and fusion on the major amblyoscope have been checked, there is often time to include numerous other orthoptic activities such as amblyopic exercises or, if the correspondence is normal, cheiroscopic work with mosaic patterns, blocks, or small models. Then, when the mother has become familiar with the many monocular

exercises for the infant squinter, she may be given work to supervise at home. These exercises may include coloring outlined pictures, tracing stencils, fitting picture-puzzle or construction blocks, stringing beads, cutting out pictures of all kinds such as paper dolls, or sewing dotted picture-cards. The last two activities of course require dull-pointed scissors with a rounded top and needles with ferruled ends. All of these exercises should be supervised to a certain extent but still allowed to remain play activities of the child.

It must be admitted that all of us are tempted constantly to try active treatment on these little squinters, who are always ready for our games, but we all know that usually even the attempt is a mistake. These infant bodies and minds are quite naturally not capable of anything but play in a very active form, and these baby eyes cannot be taught to work together in any form of play. The awakening of latent fusion requires much intense concentration, which is often a real task for the adult. To attempt such teaching with a child under four is to take the chance of creating a distaste in the infant's mind for all orthoptic exercises—a distaste that will always be remembered. Before taking such a chance, in any case, should we not ask ourselves if it is really worthwhile and will it not be better to let the infant wait for straight eyes until he is ready to coöperate without endangering his body and mind?

721 North Seventeenth Street.

THE USE OF PRISMS IN ORTHOPTICS*

CLARA BURRI, PH.D.
Chicago

I am always impressed by those who categorically can enumerate the pros and cons of a given method or procedure in orthoptics; since to me any orthoptic instrument, or method of measurement or training, must be adapted to the specific case. The goal, however, is always the same; namely, the reëducation of a disturbed visio-motor-fusion mechanism. Every patient who comes to me I look upon as an individual with a distinct problem in the use of binocular vision, and my aim is to help that individual to solve his problem, which means to teach him how to attain normal and comfortable binocular vision; or, in some cases, to attain parallelism of the visual axes if possible. I never treat or train just eyes. I always train a person. With this point of view my methods of training are very flexible and adjusted to each particular individual. Not that I do not have some general or fundamental methods and tools. I do, but I may vary them, or use them in various combinations, according to the specific problem.

In each and every case I ask myself, first of all, which method or combination of methods will bring my patient most quickly to his goal; and, of course, as each personality differs the methods of approach must vary accordingly. Since I never train a squint or a phoria or a convergence insufficiency or a divergence excess, but rather, a person with a visual problem who is to be reëducated to achieve normal binocular vision, the methods of training must always be consistent with existing laws of pedagogy. Thus the essential thing is to study each patient, to analyze the problem as it applies to him, and then to devise the system of procedures most likely to attain good results for that individual. With this point of view the methods of training may become as variable as are subjects or patients, and what may bring results in one case may give little success in another.

Thus when I was asked to prepare a paper on the use of prisms in orthoptics my first reaction was: Why not cover the whole subject of orthoptics, since prisms are used in one way or another throughout most of the field? However, this paper presents an attempt at systematizing the use of prisms and at categorizing some situations in orthoptics in which prisms are used to particular advantage. In order to do so, it is essential, perhaps, to divide the subject into: (1) the use of prisms for making diagnosis and measurements; and (2), the use of prisms for training and treatment, when these are continuously worn by the patient.

Prisms in measurements. For diagnosing and measuring muscle anomalies prisms have become one of our primary tools. An ocular-muscle examination without the use of prisms is hardly conceivable. The cover test is the quickest and one of the most accurate of all tests with which to measure deviations, and it should be in continuous use for every case and at almost every visit. The cover test not only gives accurate and quantitative measurements, but it also furnishes a quick check on the results obtained by other tests, and a quick review of a given case preliminary to each training period. The

* From the Department of Ophthalmology, Northwestern University Medical School, Northwestern University. Read at the second annual meeting of the American Association of Orthoptic Technicians, in Chicago, October 12, 1942.

cover test, carried out with the aid of either loose hand prisms or any standard prism bar or rotary prism, is the one single tool which may be considered essential for the routine examination. The test should be carried out in the primary and the six cardinal positions.

Again prisms are used in the Maddox rod test in which that amount of prism is found which gives the patient superposition of the light and Maddox streak. The numerical reading on the prism is the quantitative measure of the existing deviation, expressed in terms of prism diopters. This may be eso-, exo-, or hyperphoric; or it may be eso-, exo-, or hypertropic.

A similar test for which prisms are necessary is the diplopia test, which may be used with a red and green light or the letters "ONE." In this test, as in all others in which a deviation is measured, prisms base out are used to bring about convergence until the two images coincide, whereas in cases of divergence, prisms base in are used. These tests are particularly well adapted for testing hyperphorias, when the prisms must be placed either base down or up, depending on the case. The numerical reading of that strength of prism which gives superposition or alignment of the two images denotes the degree of the deviation; provided correspondence is normal. If fusion of the two images occurs at the expense of a false correspondence, there will be movement of the eye when covered. If the two maculas are used there will be no movement. Since this paper deals with the use of prisms as tools in orthoptics and the procedure in making a particular test is described only by way of exemplifying the use of prisms, normal correspondence should always be assumed. It must be borne in mind that abnormal correspondence often changes the procedure or, at any rate, the evaluation of the findings.

Finally, prisms are used to measure ductions. Prisms of increasing strength, either base out or base in, are placed before the patient's eyes while he is observing a small light either at 20 feet or at the reading distance. The degree of prism that induces diplopia represents the duction strength; base out measures adduction and base in abduction, base up infraduction and base down supraduction.

In cases of very high deviations the use of additional prisms may sometimes be necessary in some of the amblyoscopes and the rotoscope, but the measurements obtained in this way are usually not very reliable, because it becomes difficult to fixate with both eyes owing to the mode of construction of the instrument.

Prisms for training may be used in various ways. They become a useful tool when employed in loose-prism exercises or they may be used in connection with some instrument. If the angle of squint is over 10 degrees of convergence or divergence, prisms are frequently helpful in such instruments as the myoscope, cheiroscope, and the stereoscope. For example, in using the myoscope, the patient may be given red and green spectacles and then be made to try to fuse the two colored images into one. If the angle of squint is so large that the eyes cannot fuse these images, prisms are placed in the lens holders. These prisms must be strong enough so that the images come together within the range of fusion. When fusion amplitude increases, the prisms may be made weaker.

When the cheiroscope is used, prisms up to the strength of the squint must be placed over the lenses. The child should never be allowed to work without prisms of the proper strength; otherwise he will not be able to fuse, and will only practice alternating fixation.

Variable prisms are also useful in connection with the stereoscope, which aids in

developing amplitude of fusion; and to give abduction and adduction exercises from home training. However, before any of these instruments are used care must be taken that the patient does not suppress or alternate, and especially that normal correspondence has been well established.

In cases of squint prism exercises with loose prisms are of doubtful value. Perhaps with very intelligent and coöperative patients loose prism exercises may be prescribed for home exercises; especially in postoperative cases in which only small amounts of squint remain and in which fusion and good fusion amplitude were well developed before the operation. In this case the prism should be used base out for convergent squint, and base in for divergent squint, the strength being such as to allow the patient to fuse the two images. The prism should be held in front of one eye, and the strength gradually decreased. A prism bar of graduated prisms is useful for this type of exercise, and the training should be done at various distances from the eyes.

Even if exercises with loose prisms are of doubtful value in cases of large deviations, they are most useful in dealing with phorias, especially exophorias. It is essential that the kind of phoria be carefully diagnosed and the individual's ability to cope with that phoria. This necessitates careful measurements of the phoria and the ductions, and for these measurements prisms and a Maddox rod may be employed to great advantage. The measurements are made in the manner aforedescribed. The maximum strength of prism which the patient can overcome is the measure of his duction power. According to Dr. Berens, the normal ratio of abduction to adduction is considered to be one to three. But it is not only important to ascertain the ratio and the breaking point and that of recovery, but also to consider the relationship of the size of the duction to that of the phoria. What is frequently considered a fair average of adduction power for orthophoric eyes may be completely insufficient when a phoria is present, and by increasing the adduction reserve comfortable vision may be achieved.

For esophoria and abduction weakness prism exercises alone are frequently not very effective. However, in making the attempt, prisms base in are placed before each eye and their strength gradually increased until the maximum strength is reached. Better results will be obtained in prism training with stereoscopic exercises on one of the stereoscopes, or the rotoscope, or one of the major amblyoscopes. When the ordinary stereoscope is used it must be equipped with spring clips for the prisms. These may be increased one or two diopters at a time. This exercise is of benefit in cases wherein the phoria is small and there exists a weak abduction. When there is a fair sized esophoria that is greater for near than for distance, and greater without than with the correction (if this is hyperopic), good results may be obtained with prism and stereoptic exercises after the physician has prescribed full correction. Still better results are obtained if the patient uses some homatropine at night. This last procedure usually helps if the phoria is due to accommodative spasms.

But it is in cases of exophoria and convergence insufficiency that prism exercises may be used with best results. In this exercise one increases the ability to converge by placing the prisms base out in increasing strength. The patient begins with prisms of moderate strength, small enough so that he is well able to overcome them. The prism strength is gradually increased to a maximum degree. The exercise should be performed daily for 10 to 15 minutes. In many cases the patient will learn to overcome the effect of from 50 to 80 prism diopters. Of course fre-

quently these exercises should be supplemented by exercises on one of the major amblyoscopes, the rotoscope, or other major training instruments. These directions are not meant to imply that the exercises described are necessarily the best, nor that they should always be used, but merely indicate the various possibilities of using prisms for orthoptic training. Prism exercises frequently have no effect on the exophoria. However, since they increase the convergence and adduction power and reserve, they increase the individual's ability to cope with his phoria so that he becomes able to read and do other near work with greater comfort.

In order to determine the proper treatment for a given condition, a careful analysis of a patient's muscle imbalance must be made. One should distinguish between different types of exophorias. There is the accommodative type that is found in myopia, and essential exophoria that is apparently unrelated to refractive errors. The latter normally occurs in patients with hyperopia, and quite often the refractive error is very small. Here an insufficient convergence is present or one that is weak. Exophoria is present in presbyopic eyes, in which the accommodation becomes less as the lens becomes sclerosed. Furthermore, the use of bifocals diminishes the response required from the ciliary muscles, which diminution is followed by a corresponding relaxation of convergence. The choice of the treatment should be determined by all these factors, and after the physician has ascertained the refraction and has cleared up any problems of general health. If exercises are prescribed, loose prisms or prisms in connection with some training instrument may be used, but these should not be considered the most satisfactory methods.

In cases of vertical deviations prism training is not so satisfactory, perhaps because the average vertical muscle balance is stable, varying but little up or down. Three or four degrees is about the limit of one's supraduction or infraduction ability. Since the flexibility in vertical balance is so slight, training with prisms will accomplish but little. However, occasionally a patient is found who benefits from exercises with vertical prisms, carried out on the rotoscope or some other training instrument. Two cases in particular may be cited. In the one there was a right hyperphoria of four prism diopters. Incorporated in her glasses the patient wore prisms that corrected the hyperphoria. When wearing those glasses she had frequent diplopia and headaches. However, after receiving fusion and duction exercises on the rotoscope, during which time the strength of the correcting prisms was gradually decreased at each session, she was able to dispense with the prisms except for near work, and diplopia occurred only when she was tired.

The other patient, a dental student with an old paralysis, gradually decreased a varying left hypertropia of 8 to 13 prism diopters to a constant hyperphoria of 3 diopters. He took exercises for more than six months, and now is wearing in his correction 2 prism diopters base down before the left eye and 1 prism diopter base up before the right eye. Of course it may be suggested that his hypertropia decreased because of a gradual and natural disappearance of the paralysis. This may be true, but this condition existed for over five years previous to the treatment. Surgery had been advised but the patient preferred to try the exercises first.

Prisms for constant wear. Some authorities have advocated the use of prisms for squint. The principle is to place before the eyes a prism of such strength that rays of light entering the eyes are diverted so that diplopia is avoided, or, theoretically, so that the rays fall on the maculas and fusion is forced even though

convergence is present. These prisms are worn in addition to the ordinary glasses, usually in the form of slip-overs. The theory is that the prisms produce a desire to fuse and as fusion develops more and more strength of the prisms is continually decreased until the patient can fuse with the eyes in the primary position.

There are several arguments against such a use of prisms in cases of convergent squint. First, according to the laws of learning, we cannot assume that a patient would learn fusion merely by the presence of prisms which divert the rays of light on the two maculas. If fusion could take place so easily many would probably have never developed a squint in the first place. Furthermore, if abnormal correspondence were present, that visual habit would be more recent and stronger than macular vision. According to the laws of learning we may assume that the eyes would tend to respond in the way most strongly fixed. That this actually happens is clear when we consider how difficult it sometimes is to break up abnormal correspondence. Also in cases where surgery was performed in the absence of orthoptic training either before or after the operation, the eyes frequently revert to the old position. Learning to see binocularly follows the same laws as any other type of learning, and in all learning formal practice is necessary. Psychologists have demonstrated many times that accidental learning is poor. According to this we may well doubt that the mere providing of the correct stimulus—namely, light diverted onto the maculas of both eyes—will be able to establish the correct habit. It requires active and controlled practice. There must be conscious practice in using the maculas together. Another argument that may be presented against the constant wear of prisms is that such prisms usually induce extreme discomfort. If the deviation is great the weight of such glasses is considerable. Also dispersion of light frequently causes asthenopia and other symptoms of discomfort. Furthermore, it is important that the patient look through the optical center of his glasses, and when prisms are used the prismatic effect may easily displace that center when the glasses are not exactly centered, or when careless handling bends the frames.

Some ophthalmologists seem to prescribe prisms for constant wear for all types of phorias, particularly in exophorias and convergence insufficiency. In these cases prisms base in are ground into the lenses of the patient's glasses, and may be given to any patient, but are prescribed particularly for patients over 60 years of age. One doctor of my acquaintance prescribes as though by a fixed rule, two diopters of base in, or one diopter in each lens, in every reading segment of two and a half or more diopters.

The viewpoint adopted in this paper is that the most important muscle deviations requiring prism correction in the glasses are those in the vertical direction. A small hyperphoria of not more than two prism diopters may give an individual many distressing symptoms, and the writer has found that even when such a hyperphoria is accompanied by considerable eso- or exophoria it is frequently the hyperphoria that is the cause of the symptoms. Even with very small hyperphorias the symptoms may be great and may be the cause of defective binocular vision. Tilting of the head toward the shoulder on the side of the hyperphoria is often observed, and this phenomenon usually indicates a vertical deviation which should be corrected.

A tendency toward vertical hyperphoria is also frequent in connection with lateral squint. This vertical deviation seems to interfere with fusion, causing the eyes to alternate as soon as the two images approach the maculas. Because these images

are not vertically aligned fusion becomes very difficult, and if a tendency toward abnormal correspondence is added to this situation, superposition of the two images becomes almost impossible. A correction of the vertical deviation may in such cases mean the difference between success or failure in teaching binocular vision and normal correspondence. The development of adduction in convergence insufficiency may also be speeded up greatly if any complicating hyperphoria is first corrected.

However, the advisability of prescribing prisms for constant wear seems to be debated by several authorities on motor anomalies. Alexander Duane[1] stated that prisms when employed for constant wear are sometimes serviceable in anomalies of the individual muscles, and particularly in vertical deviations, but they may cause mischief in convergence insufficiency or convergence excess or in divergence insufficiency, by further dissociating the very functions which are to be corrected. He warns us that prisms for constant wear are merely crutches and as such should be used only temporarily. The object is to improve the binocular visual function and to integrate the eyes in such a way as to achieve good fusion and ductions and to secure comfortable binocular vision.

William Thornwall Davis[2] points out that the correction of hyperphoric errors with prisms may relieve the patient's symptoms but will not cure them; and he emphasizes the importance of cure, where it is possible, because hyperphoric errors may be a serious handicap to the individual. For example, prisms in glasses would be of little help to the many youths who wish to become aviators. The army and navy air-corps standards are high, and where the phoria is in excess of the permitted standard, entrance is not permitted. Entrance into other fields is likewise barred by muscle imbalance. The prescription of prisms in glasses for the correction of symptoms is not directly within the scope of this paper. However, since the two subjects frequently overlap, and are even confused at times, it is well to mention the problem and point out the difference between the two procedures, namely prisms for correction and prisms for the relief of symptoms.

Two very good examples for which the latter procedure is appropriate are the use of prisms in glasses for paralysis of divergence and their use for paralysis of convergence. In paralysis of convergence, prisms base in should be prescribed. In this condition the patient experiences crossed diplopia within a certain distance, usually nearer than 36 inches. The images become further separated as the object is brought closer to the eyes. The prisms are usually limited to use in near work and the amount required will be that required to superimpose the images at the reading distance. In paralysis of divergence the condition presents the opposite picture. Here binocular vision is normal from about 2 to 10 feet, outside of which diplopia exists. The double images are homonymous and become further separated the farther away the object is from the eyes. In this case prisms base out are used for distance vision and that amount of prism is provided which superimposes the images at 20 feet.

25 East Washington Street.

References

[1] Duane, A. A new classification of the motor anomalies. Ann. of Ophth., 1896, Oct.
[2] Davis, W. T. Ocular motor anomalies. The West Virginia Med. Jour., 1939, v. 35.

NOTES, CASES, INSTRUMENTS

DERMOID TUMOR OF THE SCLERA

LELAND F. CARTER, M.D.
Detroit

Orbital and epibulbar dermoid tumors are not so uncommon as to be extremely rare. While the orbital variety is the more frequent in occurrence, the epibulbar tumors are the more interesting since they afford more opportunity for observation and study. On the bulb their most frequent site is in the conjunctiva, where they are freely movable over the globe; next in frequency are those firmly fixed at the corneoscleral junction. In the literature and in the textbooks (Duke-Elder,[1] Parsons,[2] American Encyclopedia of Ophthalmology) little or no mention is made of dermoids of the sclera.

Duke-Elder[1] and Collins and Mayou[3] state that, "on the surface of the eyeball in the upper outer quadrant congenital growths are sometimes found containing bone, hyaline cartilage, or acini-tubular gland tissue resembling lacrimal gland. These growths are entirely in the conjunctiva being freely movable over the sclerotic. . . ." A tumor similar in appearance except that it had its origin in the lower temporal quadrant of the sclera is here described.

CASE REPORT

A male infant, 24 hours old, was seen in consultation. A small, round, angry-red tumor (5 by 4 by 3 mm.) was observed projecting between the lids of the right eye near the external canthus (fig. 1). It appeared to lie in a sulcus or "coloboma" of the lower lid from the inner surface of which a rather dense web fanned out to become firmly attached to the tumor. The lower cul-de-sac was uninvolved, a probe could be passed freely between the tumor and the lid. The tumor was hour-glass shaped with its base firmly attached to the sclera.

The parents were advised to postpone removal for several months, when the child could safely be given a general anesthetic. Ten days later, because of the anxiety of the parents, operation for removal of the growth was performed under local anesthesia (pontocaine, 0.5 percent; novocaine, 2 percent). Attempts to inject the scleral attachment of the tumor were unsuccessful because of the bony hardness. The surrounding bulbar conjunctiva and lid attachment were infiltrated with novocaine, and one large conjunctival vessel going to the tumor was tied off.

Fig. 1 (Carter). Dermoid tumor of the sclera in a 10-day-old infant.

The weblike connection between the lid and the tumor was severed at the lid margin, giving free access to the growth. After several attempts, the tumor was finally cut through with a pair of Stevens scissors flush with the sur-

rounding sclera. A dark bony base remained, no doubt a portion of the tumor, but no effort to remove more tissue was made for fear of entering the globe. The wound was closed by two overlapping conjunctival flaps. The rounded edges of the V-shaped "coloboma" of the lid were scarified and the defect closed by four interrupted black-silk sutures, two through the tarsal plate, one mattress suture through the lid margin, and one through the skin.

Fig. 2 (Carter). Photomicrograph of dermoid tumor of the sclera, showing epidermis, sebaceous glands, and bone.

Recovery was uneventful and the sutures were removed on the tenth postoperative day.

Microscopic examination: "The specimen (fig. 2) is skin and subcutis. The epidermis is thickened and has one area of ulceration with some associated small round and polymorphonuclear cell infiltration about the ulcer. There are sebaceous glands, an occasional one being dilated, and a few sweat glands. There are numerous groups of tubulo-alveolar glands, apparently well differentiated lacrimal glands. In the center of the piece there is a mass of bone and osteoid tissue in association with a proliferation of anaplastic cells which have hyperchromic nuclei, little cytoplasm, and which are quite variable in shape. This tumor is malignant.

"Diagnosis: Mixed lacrimal-gland tumor with malignant changes."

Drs. Parker Heath and Plynn Morris were of the opinion that considering the

age of the patient the character of the cells was not unusual. In their opinion the tumor was a nonmalignant dermoid.

Comment

Dermoids of this character have generally been classified as teratomas, but Collins and Mayou choose to regard them as an atavism of tissue, since the corium of the skin in some animals is capable of producing cartilage and bone in the form of the exoskeleton. To them the Verneuil theory of dermal inclusion seems the most logical. Parsons[2] also expressed it as his opinion that this theory was strongly supported by the site of the election of these tumors, situated as they are at or near the fetal clefts or sutures. That a certain number of them represent the residue of amniotic bands, Duke-Elder[1] is "tolerably certain."

In the case here under consideration Herrenschwand's hypothesis of an organic adhesion of the lid margin to the surface of the globe as a possible cause is in part at least borne out by the rather heavy bridge of tissue spanning the cul-de-sac from lid margin to tumor. On the other hand, the fact that the lid defect was in such intimate contact with the tumor lends ample support to the dermal-inclusion theory. The fact remains, however, that there was in appearance no apparent loss of lid substance and that upon repair, as in any simple laceration, there was no shortening of the lid.

Thus on the basis of these findings one might assume that an amniotic band or bands gave rise not only to the tumor but were instrumental in a mechanical way in bringing about the lid defect.

Twelve months following removal of the growth (fig. 3) there is a moderate thickening of the conjunctiva in the lower temporal quadrant but no evidence of recurrence. The fundus is entirely normal except for some grayish discoloration, but there is no elevation of the choroid at the extreme periphery in the lower temporal region. There are no colobomata of the iris or choroid nor other congenital anomalies. The left eye is normal.

Fig. 3 (Carter). Appearance of eye 12 months after removal of tumor.

A somewhat similar dermoid cyst, the base of which was "substituted for the conjunctiva, sclera, and choroid coats of the eye," was described by Miller.[4] An interesting case report was also published by Calhoun[5] of a dermoid tumor of the cornea and conjunctiva associated with a scleral ectasia in the lower nasal quadrant.

SUMMARY

An unusual case of congenital epibulbar tumor is described which involves the conjunctival, scleral, and probably choroidal coats of the eye. The microscopic section showed the presence of cutaneous and subcutaneous tissue, sebaceous and sweat glands, numerous groups of tubulo-alveolar (lacrimal) glands, and a central mass of bone tissue. There is associated with this a "coloboma" of the lower lid.

Three possible causes of such a tumor are described, but no definite conclusion has been drawn in this instance.

613 David Whitney Building.

REFERENCES

[1] Duke-Elder. Textbook of ophthalmology. St. Louis, C. V. Mosby Co., 1938, v. 2, pp. 1407-1412. Philadelphia.
[2] Parsons. The pathology of the eye, v. 1, pt. 1, pp. 132-138.
[3] Collins and Mayou. Pathology and bacteriology of the eye. Philadelphia, P. Blakiston's Son and Co., 1925, pp. 141-143.
[4] Miller, I. M. Amer. Jour. Ophth., 1924, v. 7, Sept., pp. 703-704.
[5] Calhoun, F. P. Amer. Jour. Ophth., 1924, v. 7, Sept., pp. 669-670.

THE TECHNIQUE OF TONOMETRY AND CARE OF TONOMETERS

MARK J. SCHOENBERG, M.D.
New York

Possession of an accurate tonometer, proper care of the instrument, correct technique in using it, and complete recording of the tonometric findings are the four requisites of reliable tonometry.

Since neither textbooks nor ophthalmologic magazines contain explicit instructions concerning the subject mentioned in the title of this paper, the author has deemed advisable to submit the rules followed at the Glaucoma Clinic of the Manhattan Eye and Ear Hospital of New York.*

The tonometer. The instrument must be reliable, certified as to accuracy of performance by a recognized "checking station."

The care of the instrument. The tonometer must be kept *clean* and *aseptic*—the same rule applies to the box in which it is deposited.

Cleansing is effected by wiping the interior of the cylinder, the foot plate, the foot of the plunger, and the testing block—first with a piece of cotton wound tightly around a toothpick and immersed in 50-percent grain alcohol, then rinsed off with sterile water and dried with cotton.

This procedure must be followed after each tonometric examination and, again, once before the day's consultation hours. (Now that epidemic keratoconjunctivitis is prevalent in many sections of the country, it is timely to warn against infractions of the rules of asepsis.)

If available a separate tonometer should be used for septic cases; if not available, the tonometer must be cleansed with great care after such measurements; if only the one eye is infected, the tonometer should be applied first on the clean eye.

Technique of tonometry. (a) The examiner washes his hands with soap and water before and after each tonometry. (b) The instrument is to be manipulated gently by holding the handle between the thumb and adjoining two fingers—the

* The Glaucoma Clinic at the Manhattan Eye and Ear Hospital of New York is sponsored by the National Society for the Prevention of Blindness.

pointer and hammer are not to be handled. (c) Check the tonometer on the testing block to see whether the pointer stops at the zero division. (d) In applying the instrument, the examiner faces it squarely so as to avoid a parallactic error in reading the scale. (e) He holds the instrument *vertically,* so that the axis of the patient's eyeball and that of the tonometer are in one straight line. (f) The foot plate must cover as exactly as possible the center of the cornea. (g) The eyelids are gently separated without the exertion of any pressure on the eyeball. (h) If the eyeball is deeply set in the orbit, it is often necessary to obtain the help of an assistant, who pulls down gently the lower lid while the examiner retracts the upper lid, just enough to allow the foot plate to cover the central portion of the cornea. (i) The entire procedure is to be made in a calm and gentle manner, so that the patient should be given a chance to coöperate without becoming anxious or tense. (j) If the instrument and the technique are correct and if the patient is relaxed and coöperative, two or three consecutive applications (of the tonometer) will give the same reading or a difference not greater than one-half a division. (k) Avoid covering the patient's second eye in the process of keeping the lids apart, or with the hand holding the tonometer.

The patient is placed on a table or reclining chair with the face directed straight up; the collar is loosened to prevent pressure on the jugular vein, thereby increasing the intraocular pressure.

One drop of a weak anesthetic (pontocaine 0.25 to 0.50 percent or butyn 0.50 percent) is instilled twice at an interval of 10 seconds (preferably above the limbus corneae while the patient is looking down). Ready, within one minute, the patient is told to direct his eyes straight up to a target on the ceiling or preferably to his own hand, held in a suitable position indicated by the examiner. Any deviation from the straight-up position leads to errors in measurement. In some apprehensive patients, coöperation and steadiness of gaze are secured by asking the patient to watch the target, whether it changes color during the examination ("whether it is becoming blue or green"). This induces the patient to concentrate on something else than on his own fear.

Unruly or overanxious patients have to be carefully trained before the tonometric reading can be relied upon; this applies especially to one-eyed patients.

At the conclusion of the procedure, one drop of 10-percent argyrol is instilled in each conjunctival sac and the eyes are irrigated with salt or boracic-acid solution —just enough to clear the eye of some of the argyrol. (In very rare cases, patients are allergic to argyrol.)

Recording the tonometric findings. The following data must be recorded: (a) date and hour of day; (b) the reading on the scale; (c) the number of hours or days since last instillation of the miotic; (d) the name and dosage of the drug used; (e) the strength of the miotic and frequency of instillations.

667 Madison Avenue.

A NEW SUTURE FOR USE IN MUSCLE-RECESSION OPERATIONS

CLARANCE B. FOSTER, CAPT. (M.C.), A.U.S.
New York

For some time, Wendell Hughes of New York and others have used, in children, a modification of the Jamison technique. A double-armed suture is placed vertically, passing through the superficial layers of sclera, crosswise to the direction of the fibers, at the point of selection. The suture is passed through correspond-

ing points in the muscle and tied in a surgeon's knot.

The writer has used this method in adults with uniformly good results.

A further simplification and extension of the modification is now suggested.

Before the tendon is cut, and after the muscle hook has isolated the tendon, and is still in place, a single-armed suture on a cutting-edge needle is passed through the tendon, about one fourth of the distance from one edge, from scleral side toward conjunctival side, and back through the tendon toward the sclera, at a corresponding point one fourth of the distance in from the opposite edge. This may sound complicated, however it is extremely simple. Lifting the muscle hook like a lever exposes the required edge of tendon and provides plenty of space for the passing of the needle. The tendon can then be severed from its scleral attachment, the suture being retained as a stay suture. At the point of selection, the scleral bite crosswise to the scleral fibers can be made, going, of course, in the direction opposite to that of the initial passage through the tendon. Upon emergence from the scleral fibers, the suture is passed from scleral side to conjunctival side through the tendon, just inside the first entry. The suture can then be tied without too much tension. The tendon is well flattened down and in good approximation to the sclera.

This method has the advantage of using only one suture, and that of a saving of tendon. (Many small recessions are neutralized by the shortening involved in placing sutures behind a muscle clamp.) It can also be used in cases where a muscle clamp is not available. The value of such a procedure in war surgery is obvious.

52d Station Hospital.

SOCIETY PROCEEDINCS

EDITED BY DR. DONALD J. LYLE

CHICAGO OPHTHALMOLOGICAL SOCIETY
CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

JOINT MEETING

February 8, 1943

LOUIS G. HOFFMAN, *president,*
Chicago Ophthalmological Society
G. HENRY MUNDT, *president,*
Chicago Laryngological and Otological Society

SYMPOSIUM ON HEAD AND FACE PAINS
(Abstract of Proceedings)

INTERNAL MEDICINE

DR. G. W. SCUPHAM. The lay public has been so well educated to the fact that eye strain and eye diseases are frequent causes of headache that they will go to the ophthalmologist, and the otologist has his turn because of the belief that sinus headaches are common. It is a difficult problem because of the multiplicity of causes of which headache is the outstanding symptom. From the standpoint of the internist, not only is the diagnosis difficult, but the therapeutic problem is often more so. If we limit ourselves to any one of the fields of medicine in which headache is important we are likely to fail to arrive at a conclusion in a particular case.

A number of mechanisms may result in various types of headache, but so far as head and face pains are concerned, these are not so important. Pain may be the result of local diseases or local reflex phenomena. Some blood-vessel disorders are characterized by pain. Arteritis of the temporal vessels does result in a throbbing, burning type of pain localized in the region of these vessels, often associated with evidence of an inflammatory process. Herpes zoster is a fairly common cause of head pain. Tumors of the scalp and skull, multiple myeloma, and osteitis of the skull might also be mentioned.

Headaches are more important to the internist than head and face pains. The character of the pain seems to be different. Headache arises within the head, whereas head and face pains are more or less superficial. Headache is extremely variable in different individuals. It is uncommon in children, and in aged individuals it is less common than in middle adult life.

The majority of headaches are those which are merely part of the general symptomatology of the disease picture. In almost all acute infections headache is an important symptom. Typhoid fever is often characterized by long-continued headache; influenza and acute respiratory infections by headache at the onset. An example of toxic headache is that resulting from sulfathiazole; alcohol is also a cause of this type; carbon-monoxide poisoning is also toxic, but often results in structural changes. In acute nephritis, headache, associated with nausea, is an important symptom of the onset of the disease. It is in part toxic but there may be a rise in intracranial tension; in the uremic phase of chronic nephritis the headache is probably toxic. Headache is a common and extremely important symptom in chronic malaria, is persistent and severe, and disappears with control of the disease.

Hypertension is the major vascular disease in which headache is important. The

inhalation of amyl nitrite results in headache and fall of blood pressure. This is an interesting contrast in the mechanism of headache, as in persons having hypertension a fall in blood pressure often overcomes the headache. Many with severe hypertension never have headache, and many with moderate hypertension complain of severe throbbing headache, often continuing for days without marked exacerbation of the blood pressure. In general, patients whose hypertension is in the malignant stage are subject to severe headache, probably because there is marked vasoconstriction of the arterioles of the central nervous system.

The headache that resembles the type caused by injection of histamine, may be confused with migraine because it is often unilateral. There is no history of familial headache. The headache often begins at night. There is often flushing of the head and face on that side, tenderness of the temple and over the carotid artery. Pressure on the carotid may result in relief. A small amount of histamine injected subcutaneously can initiate an attack. The recognition of this type is important because relief may be obtained by the therapeutic administration of histamine.

There are a great variety of rheumatic headaches, from that of acute rheumatic fever, which may be mistaken for brain tumor, to a mild occipital headache of chronic nature. Perhaps the common type is associated with fibrositis and myositis as well as with cervical osteoarthritis. Such headaches occur with increased tension of the muscles of the spine and neck.

Reflex headache is more difficult to differentiate. It is often associated with disease of the pelvic structures. Constipation is said to cause headache reflexly and evacuation of the lower bowel brings relief.

The typical features of migraine headache are well known. The family history is so common that if we do not find it we doubt the diagnosis. There is a slow development of headache with nausea and vomiting, relief and sleep, often preceded by aura. It is extremely variable in some individuals, and it sometimes disappears at or near the menopause. Another striking feature is the association with the menstrual cycle. Migraine may be precipitated by a wide variety of factors; food allergy, emotional states, certain endocrine abnormalities, particularly the gonadotropic factor, fatigue, infection, and many others. It is commonly thought to be incurable, but much can be done if the problem is studied. Treatment should be directed toward elimination of the exciting factors when possible.

NEUROLOGY

Dr. Lewis J. Pollock. When a stimulus is applied to the skin or mucous membrane on the exterior of the body, sensation is transmitted by the sensory fibers of the craniospinal nerves and reaches consciousness directly through their appropriate sensory tracts. Thus, inflammatory and other pathologic processes of the skin and mucous membrane produce stimuli carried by ordinary sensory cutaneous nerves and are localized by one to the area stimulated.

When the subcutaneous areas are stimulated, sensation is transmitted in two ways: first, by the ordinary sensory components of craniospinal nerves carrying deep sensibility—in the case of the head and face, by way of the sensory components of the 3d, 4th, 5th, 6th, 7th, 9th, 10th, 11th, and 12th cranial nerves; second by a more complicated mechanism producing referred pain—as an example the shoulder-tip pain occurring when the central portion of the diaphragm is stimulated by diaphragmatic pleurisy. The impulse travels by way of the phrenic nerve through the posterior roots of the 4th

and 5th cervical nerves to the spinal cord, there making a synapse with the descending short fiber tracts to the points of origin of the cervical sympathetics, then through the preganglionic and postganglionic fibers to the skin, where a chemical change is produced stimulating the sensory end organs, and sensation is then carried by the sensory spinal nerves into the spinal cord and by its appropriate sensory tract into consciousness. The location of pain is then referred to the skin over the shoulder tip.

The same mechanism applies to the head and face. In the case of vascular headaches, and perhaps migraine, stimuli occur as the result of dilatation of the blood vessels; impulses travel by way of the nervi vasculorum originating in the cranial nerves, perhaps also upper cervical, and find their way into the brain stem and medulla; there a synapse with the short descending pathway of the cervical sympathetics occurs; impulses are sent over the postganglionic fibers to the skin, where again chemical changes produce a stimulus in that area, and impulses carried by the particular sensory nerve involved, chiefly the fifth but also others, enter the brain stem. This holds true also of distention of other subcutaneous tissue as well as traction on the meninges and dural sinuses. This can be proved by experiments in which the pain ensuing from faradic stimulation of the superior cervical ganglion can be stopped by section of the sensory root of the fifth nerve. The pain cannot be stopped by severing the preganglionic fibers when the appropriate anterior roots are severed or by cutting the cervical sympathetic chain below the superior cervical ganglion.

Usually head pain does not occur as the principal symptom of intracranial disease; although headache may be the presenting symptom, it is not the sole symptom in intracranial tumors. Diplopia, hemiparesis, facial paresis, ataxia, loss of visual acuity, vomiting, and mental disturbances are always at times associated with the headache. Headache when present is associated with vomiting which may be projectile in character and usually does not lead to alleviation of pain. Here the headache is sharp and severe and continuous; it may be a throbbing, stabbing, boring pain. It is often brought on by sudden change of posture and may disappear by changing the position of the head; in intraventricular and infratentorial tumors, the patient may hold his head in a position which tends to lessen the possibility of occurrence of pain. It may be associated with suboccipital tenderness and rigidity of the neck. It is increased on jolting and straining and, with sudden exacerbation of pain, vomiting may occur.

The location of pain is not characteristic, nor is it an index of localization of the tumor. The character of the tumor may influence the type of headache and its severity. Rapidly growing tumors are associated with more headaches; hard tumors cause more pain; circumscribed tumors produce pain of greater intensity than do infiltrating tumors. Tenderness to percussion of the skull at times serves to bring out an area of circumscribed tenderness which may indicate intracranial disease.

Associated signs and symptoms lead to the diagnosis. Hemiplegia of sudden onset points to hemorrhage, thrombus, and embolism; the sudden onset of excruciating suboccipital pain with loss of consciousness, rarely hemiplegia, and rigidity of the neck, characterize subarachnoid hemorrhage, usually due to ruptured cerebral aneurysm. Inflammation of the leptomeninges, alone or with encephalitis, is associated with rigidity of the neck, Kernig's sign, and Brudzinski's sign; and evidence of inflammatory disease is found on examination of the cerebrospinal fluid.

Serous meningitis with paroxysmal hydrocephalus is characterized by the occurrence of definite attacks of headache, vomiting, sometimes choked discs, and signs of interference with cerebral function. Such attacks, after a relatively short course, may disappear and a considerable time elapse before another occurs. In such cases one may often obtain a history of another phenomenon of anaphylaxis, as angioneurotic edema or Menière's syndrome.

Most cerebral diseases produce interference with function of motion, sensation, tone, synergy, speech, reflexes, and cranial nerves. No more than a consistent difference in the activity of deep reflexes upon the two sides may be noted, but when this is combined with diminished superficial reflexes on the same side it is a strong indication of organic disease. Changes in the visual field may be found long preceding change in the optic nerve, especially in temporal-lobe lesions, wherein a quadrantic anopsia and a headache may be the sole symptoms of tumor. Whether encephalitis uncomplicated by meningitis can produce headache cannot be stated. Many of the encephalitides are associated with meningitis, and in lead encephalopathy a marked change in renal function may be present, which in itself may produce marked headache.

In a very large number of patients suffering from headache careful search reveals no organic disease. Such headaches may be termed "psychogenic," or, perhaps better, headaches associated with neurosis. Usually the patient does not mean that he has pain in the head when he complains of headache. It is a feeling sometimes describable, at times not; always it is "intense," "terrible," "horrible," "splitting," "unbearable." It is the hyperbole and exaggeration which point to the condition. The sensations complained of are also myriad. The so-called pain may involve any part of the head, and shifts from day to day or from hour to hour. It is continuous, is usually unaffected by weather or by ordinary changes in position. It is not accompanied by nausea or vomiting. It is always increased by making decisions and always by work. It is relieved by reassurance, whether by a physician's statement or by favorable observations by the patient. If the patient becomes interested in any pursuit, so long as the interest lasts the headache will abate; it is abated by any new treatment or doctrine. It is worse in crowds, and is increased by excitement, disputes, quarrels, and domestic, social, and business conflicts.

As is the case with sensations relative to phobias relating to the heart or stomach, so with this headache. A real pain or discomfort of organic origin may often have preceded it; it may follow a migraine, an indurative, ocular, or sinus headache, or may coexist with them. Prominent among the symptoms are depression, abulia, inattention, difficulty in concentration, forgetfulness, introspection, anxiety, fears, indecision, fatigue, and insomnia. It is important not only to distinguish these headaches from organic headaches, but it must be remembered that they may be the beginning of a more serious condition, a beginning melancholia, dementia praecox, or dementia paralytica.

Often pain in the head or certain types of headache are classified as neuralgia; namely, auriculotemporal, occipital, and so on, neuralgia. This I believe is a mistake. If we accept trifacial neuralgia as characteristic of the neuralgias, then sudden paroxysms of severe pain in the distribution of the sensory supply of the nerve, lasting only a fraction of a second, and brought about by touching a

trigger or algigenetic zone, are seen only in relation to the trigeminal or glossopharyngeal nerve.

The characteristic neuralgia affecting the trigeminal nerve is observed usually when no underlying pathology can be demonstrated even when the gasserian ganglion is inspected. At times it occurs in relation to tumor of the gasserian ganglion, angle tumors rarely, and multiple sclerosis. It is not related to any other extraneural disease, as disease of the sinuses, teeth, or other structures of the head and face. The characteristics of the pain are unique. The pains are always referred to the same zone, supraorbital, infraorbital, or mental, or two or all of the branches. They may occur seemingly spontaneously but can always be brought on by touching the area which is the trigger zone, or by movements stimulating such a zone—talking, chewing, swallowing. The area stimulated is not always the area in which pain ensues. The pains are very severe, sharp, shooting, electriclike, last but a few seconds, or may consist of a succession of shorter ones. There is no relief other than interruption of the sensory fibers of the fifth nerve.

ALLERGY

Dr. Samuel M. Feinberg. From the standpoint of allergy there are two main types of headaches: the classical migraine and the simple but frequently recurring headaches. We believe there is no real etiologic distinction between the types; the migrainous are merely a more severe form. We have observed many patients whose ordinary recurrent headaches go on into migraine, and other patients whose headaches vary from the simple to the typical migraine at different times.

What is the mechanism of migraine? It has been demonstrated that angioneurotic edema produces cerebral symptoms and that cerebral vessels have a vasomotor control. Several years ago a young woman who had violent migrainous symptoms was finally operated on for suspected brain tumor. Marked edema of the brain was noted but no tumor was found. Following the operation she had a defect in the skull. It was now observed that the onset of a migrainous attack was initiated by a preliminary pallor of the face and depression of the skull area. As the attack progressed there developed definite bulging of the decompressed area. Allergy studies showed definite clinical sensitivity to specific foods and the latter precipitated the headaches and the bulging of the defect. It would appear, therefore, that the mechanism of migraine is probably a cerebral angiospasm followed by vasodilation and increased intracranial pressure.

What evidence have we for the allergic basis for headaches? Dr. Scupham has already mentioned the great tendency to heredity in migraine. The majority of patients give a family history of migraine, but a large percentage also give a history of other allergy in the family. It is generally overlooked that in allergy not only is there a general tendency to the inheritance of allergy but a special tendency to have certain allergic manifestations predominating in the family. Antecedents with urticaria are more apt to have offspring with urticaria than other allergy; antecedents with migraine are more likely to have offspring with that disease than any other allergic manifestations. The fact that migrainous attacks are frequently precipitated by nonallergic factors such as fatigue, constipation, excitement, weather changes, and endocrine cyclic phenomena does not detract from the allergic concept of the disease. We know that such precipitating causes can be instrumental in being the "trigger" to other

allergic phenomena such as asthma, rhinitis, and urticaria. It is true that only a small proportion, perhaps a fourth, respond to skin tests, and yet we find that in some of the other allergic manifestations, such as urticaria and gastrointestinal allergy, the incidence of skin reactions is also small. By clinical means and observation we can assure ourselves that many patients have their headaches from foods in spite of negative skin reactions.

By the use of skin tests, simple observation, diary records, and restricted diets we can usually make a diagnosis. We may say conservatively that in half of these cases allergy can be demonstrated; as to the others we believe no satisfactory explanation exists at present. The majority of the allergic patients are sensitive to foods, the most common of which are wheat, milk, cocoa, peas and beans, potato, pork, egg, and onion. It should not be forgotten that inhalant allergens may also cause migraine.

ENDOCRINOLOGY

Dr. Hugo R. Rony. It may be expected that endocrine glands play some role in the etiology of headache for three reasons: (1) Two glands, the hypophysis and the pineal gland, are located in the intracranial cavity; (2) hormones are known to affect secretion of the cerebrospinal fluid as well as retention of water in various tissues, including possibly the meninges and brain; (3) glands participate in a number of factors known to be concerned in the production of headache —control of blood pressure, certain metabolic processes, the autonomic nervous system, the level of sugar, calcium, sodium, and other components in the blood.

It is difficult to obtain reliable statistical data in regard to the frequency of endocrine headache, inasmuch as the diagnosis of endocrine disturbance is sometimes a matter of opinion. Further, even in the presence of definite endocrine disturbance, its role in the production of the headache in a given case may be open to question. It is probably safe to say, however, that the number of cephalalgias in which glandular disturbance is a contributory factor is quite great, whereas true or primary endocrine headache is relatively rare.

In tumors and hyperplasias of the hypophysis headache is a prominent symptom; it is more or less constant, bitemporal, or occipital. It is believed to be due to distention of or pressure upon the capsule of the gland or the dural diaphragm above the gland. Eosinophilic, basophilic, or chromophobe tumors may produce this headache regardless of the functional state—deficiency or hyperactivity—of the gland. Removal of the tumor or deep X-ray therapy may completely relieve the headache. There is some evidence that the headaches of pregnancy, artificial or natural menopause, and so-called premenstrual headaches may be of pituitary origin, probably due to mechanical effects of the hyperplasia of the gland known to occur in these conditions. Such headaches may be successfully treated by large doses of estrogenic hormone preparations which are known to exert an inhibitory effect on pituitary hyperplasia.

Tumors of the pineal gland may produce severe headache of the type seen in brain tumors, especially if they lead to compression of the Sylvian duct and cause internal hydrocephalus with consecutive increase of intracranial pressure.

Headache is not infrequent in disturbances of the thyroid gland, either hyper- or hypothyroidism. Such headaches are usually mild with no characteristic localization. That they are related to the thyroid disturbance is shown by the fact that they are relieved by specific therapy, ad-

ministration of thyroid substance in hypothyroidism, or thyroidectomy in hyperthyroidism.

Headache is a prominent symptom of Addison's disease or other forms of adrenal insufficiency, and may be relieved by administration of adrenal cortex extract. In hyperinsulinism, headache may be an element of the hypoglycemic syndrome. Characteristic of this headache is its onset when the stomach is empty, with prompt relief after food intake. Hyperglycemia, on the other hand, does not in itself produce headache; however, headache frequently accompanies diabetic acidosis, and severe headache in the uncontrolled diabetic may be a danger signal of impending coma.

Headache in various conditions of doubtful glandular etiology may present a considerable diagnostic and therapeutic problem. Thus it may be difficult to decide whether headache in patients with a hypotension-fatigue syndrome, certain ovarian disturbances, or male climacterium, and the like, is of glandular or psychogenic origin. Even therapeutic success with specific glandular preparations may not answer the question of the nature of the headache, in view of the possible suggestive effect of treatment. In certain forms of obesity, especially when accompanied by hyperostosis frontalis interna (Morel's syndrome) or by painful lipomatosis (Dercum's disease) headache may be a prominent complaint. Reduction of weight in such cases is usually followed by more or less complete relief of the headache, probably because of the lowering of blood pressure and lessening of water retention as a result of loss of weight.

OPHTHALMOLOGY

DR. BEULAH CUSHMAN. Patients with headache come to the ophthalmologist sooner or later, as it has become almost a maxim that errors of refraction and muscle balance cause headache and asthenopia, and this belief is fostered by advertisements. The eye is particularly sensitive to neurosis since it is a highly specialized organ.

Of the 12 cranial nerves 4 go directly to the eye and 3 have distributions to the eye. The extensive distribution of the fifth nerve from the midbrain down to the second cervical level and the numerous interrelationships which it forms with the nuclei of origin of all the other cranial nerves, constitutes one reason why headaches so frequently result from many different causes and are so vague in distribution. The association of the trigeminus with the vagus accounts for the vomiting which accompanies acute glaucoma. This also explains the relief of dyspepsia obtained by proper correction of errors of refraction and muscle balance.

Ocular headaches have been divided into three groups: those due to retinal irritation, those due to organic disease, and those due to eyestrain.

Retinal irritation may follow glare with any sudden change of environment from a dull to a bright light, or with the use of a torch in industry. The range of sensitivity varies with the individual, partly according to the amount of pigment in the retinal pigment layer and choroid. Ophthalmic migraine, a rare condition, may be precipitated by glare, and is the type which has been described as associated with palsy of an ocular nerve, generally the third; at first the palsy is temporary, later becoming permanent. The pain is diffuse, beginning in the temporal region and spreading to the occiput. Pain and nausea always precede the paralysis. The pain in ophthalmic migraine is usually on the same side as the hemianopia, whereas in typical mi-

graine the pain is on the opposite side. The paralysis, which may involve the iris and muscle of accommodation, usually passes off in the first attack. The attacks occur only a few times during the patient's life, but each attack may continue for days, weeks, or months. The involvement of the oculomotor nerve has been constant in all published cases, but the pathologic pictures show no uniformity.

Organic diseases produce ocular pain according to the degree of abnormality of the ocular nerves and muscles. Foreign-body sensation and discharge are present with inflammation of the conjunctiva, cornea, and sclera.

Ciliary neuralgia is the type of pain present with iritis and cyclitis, is usually worse at night, and varies in intensity. Neuralgia of the eye has also been described as pain on movement of the eye and tenderness of the ocular muscles after exposure to cold air. Closely allied to this condition is neuralgia of the forehead. The symptoms may be associated with transient paresis, which responds to heat and salicylates.

The pain in acute glaucoma extends along the distribution of the fifth nerve. In cases of chronic glaucoma and secondary glaucoma there may be neuralgic type of pain referred to the face, cheek, and temporal areas, often mistaken for sinus infection until the vision becomes affected.

Retrobulbar neuritis produces a pain deep in the orbit, increased upon rotation of the eye; this pain may be present several days before blurred vision develops.

Pain in proptosis, as found in thyrotoxicosis, orbital tumor, and cellulitis, varies according to the degree of distortion of the ocular muscles and nerves and exposure of the cornea.

A refractive defect present for many years is unlikely suddenly to exert a malign influence according to Butler, unless some other factor is added—overstudy, work under unfavorable conditions, debilitating illness, and psychic disturbances. Headaches of recent origin should be due to a recent cause.

Ophthalmic headaches are presumed to be due to overwork of the muscles of accommodation and fixation; the height of pain is reached late in the day and disappears after rest. The infrequency of headaches in persons with monocular vision is said to be due to absence of effort made for fusion through complex actions. Overaction of the ciliary muscles controlling accommodation producing fatigue and spasm may be followed by headache. Refractive errors predispose to headache, as does muscle imbalance. The insertion of the frontalis muscle into the upper lid of each eye participates in the contraction of the orbicularis palpebralis; the frontalis on contraction pulls on the epicranial fascia, and this in turn pulls on the occipital muscles causing fatigue and spasm.

Headaches of ocular origin are probably most often found in the presbyopic who have a receding near point. Many persons complain of ocular discomfort, who have high degrees of hypermetropia and astigmatism or heterophoria, which had caused no previous handicap. Aniseikonia is a cause of discomfort in patients with unrelieved symptoms of asthenopia or photophobia. This may be suspected in anisometropia in unequal accommodation with lenses of unequal curve or thickness, space-perception anomalies; and in the patient with good vision in each eye who prefers to use one eye only and occasionally in a patient with reading disabilities or migraine.

OTOLARYNGOLOGY

DR. ARTHUR W. PROETZ. Reliable statistics indicate that probably less than 5 percent of headaches are due to sinus

trouble. Undue emphasis has been given to sinus headache by the public, which has been recently plied with semitechnical literature on sinusitis while being kept in ignorance of the other 30 causes of headache. The physician must guard against accepting the patient's diagnosis merely because some sinus infection is present. Many people have headache; many people have sinus infection; the two are often not related.

While the mechanism of headache is not known, much light has been thrown upon it in recent years. It is usually associated with stretching or distortion of certain definite portions of the meninges or with the dilatation of certain arteries, notably the middle meningeal. The sinuses are closely related to these structures. Congestion and inflammation in the sinuses may produce typical headache.

However, these disurbances may not be peculiar to the sinuses themselves but may be of a vascular nature and result from conditions elsewhere in the body. It is my impression that there are people who are headache subjects; that is, that many stimuli of various types produce vasomotor disturbances which set up the headache mechanism and produce the symptom typical of the individual.

Mere stretching of the nasal mucosa does not produce pain. A number of cases have now been observed of sudden edema stretching the sinus lining to 10 times its normal thickness without inducing any subjective symptoms. It does not follow that stretching of the periosteum would be painless. This may be analogous to a diffuse otitis externa, which is exceedingly painful.

The existence of a "vacuum" pain of any duration is open to doubt. I can find no evidence in the literature of any experimental proof that it exists. No physiologist whom I have consulted is willing to say that the sinus mucosa absorbs oxygen. It is pointed out that a system of tissues may react specifically in absorption or secretion of a gas. If no selective action exists and only the usual exchange of gases occurs in a sinus, then as much carbon dioxide must be given off by the membrane as the oxygen which it absorbs, and since carbon dioxide has a slightly greater volume than oxygen, a *positive* pressure must result if the ostium is closed.

Another mechanism may be at work which I have not seen described: sudden engorgement or sudden collapse of a sinus membrane in the presence of a closed ostium may conceivably produce positive or negative pressures. Whether these produce headache I do not know. In certain individuals slight exhaustion of air from the nasal fossae does produce headache.

Observation of pain in cases of known acute sinus activity, namely, acute empyema of the frontal and maxillary sinuses, leads to the conclusion that pressure *on an acutely inflamed area* causes severe pain. When the pressure is relieved the character of the pain changes. Although the mucosa is still acutely sore, the pain in no way resembles a so-called sinus headache as we understand it. It is not gone in half an hour. We are led to the conclusion that sinus headaches which come and go are the result of vascular disturbances (in or out of the sinus) and not of a fixed pathologic change.

Some nasal headaches can be demonstrated to result from the impaction of jets of air during inspiration against the nasal ganglion or its tributaries. These jets are the result of constricted airways. The direction is determined by the topography of the nose and its irregularities.

It is difficult to obtain a balanced viewpoint from the literature on headache. Many authors are themselves victims of the malady and their accounts and inter-

pretations are often highly tinged with their own experiences. This is entirely understandable, since a symptom so completely subjective can in any case be best studied by its victims.

There remains the problem of practical approach. Anyone seeing many headache patients (and who does not) must be struck by the fact that these people are prone to make their own diagnoses, which lead them to the rhinologist or the ophthalmologist or the dentist or the allergist or, what is more likely, to all of them in succession. They are commonly told, "It isn't your eyes, it must be your sinuses," or "It isn't your sinuses, it must be your teeth." They are seldom told, "Your headache is due to one or two or more of some 30 possible causes and we will try to find out which one it is."

In my own hands, the most direct method of arriving at some indication of the nature of a headache is a complete history both of the headache from the standpoint of the attack and of the background, and of the patient in general. A complete printed questionnaire helps greatly to visualize the case as a whole and to evaluate it. Important also is a complete daily record of the patient's contacts, diet, and activities. Again, a prepared chart with which I am thoroughly familiar has many advantages over a haphazard diary or even a carefully recorded diary according to some system devised by the patient. Since the important points in such a diary are often the minute details, standardized records are of great help.

(Copies of the forms used were projected on the screen.)

In the treatment of vascular headache and, in fact, of some others, to afford relief while investigation is going on, I find ephedrine with one of the barbiturates, by mouth, very useful.

I may also add that many headaches having the characteristics of sinus pains disappear on the administration of thyroid extract. A low basal metabolic rate is not always typical in these patients. I have commonly noted improvement on giving thyroid even when the rate was +5 or more.

Discussion. Dr. Peter Bassoe: It might have been well if someone had discussed recent research on the mechanism of headache. At the last meeting of the Chicago Neurological Society, Dr. Harold Wolff reported on the work by his group in New York. They have made innumerable experiments on people whose heads had been opened, and determined that the most sensitive areas are near blood vessels, which is analogous to what is seen in the peripheral nerves. The median and sciatic nerves with their arteries surrounded by nerve fibers are the areas most frequently affected by causalgia.

One speaker is missing on this program; no one discussed teeth. Even in this enlightened age a great many people have been deprived of teeth without good reason. One striking example was a woman with beautiful teeth, who had pain which she localized in a couple of teeth. These were found to be normal upon removal, but then the pain shifted to other teeth, which were also removed, until she had lost many teeth at the time she came under observation. On questioning it was found that she was in a disagreeable situation from which she could see no escape. It was possible to point out to her how this could be straightened out and the pain disappeared and the rest of her teeth remained.

Dr. Feinberg's remarks about edema and allergic conditions causing pain are interesting. One patient developed a syndrome of brain tumor, choked disc, vomiting, severe headache, but no localized signs. A decompression was made and he

was well and had no further symptoms. No tumor was found. He moved to another part of the country and then developed typical angioneurotic edema. Undoubtedly his symptoms of brain tumor were due to edema of the brain.

Another patient, a woman, had sudden severe brain headache, aphasia, and hemiplegia. She had been taking care of a child with scarlet fever, and brain abscess was suspected. She went into coma. The brain was explored and nothing but edema was found. She recovered, but temporary aphasia recurred at each menstrual period, so this was nothing but a severe menstrual edema.

Dr. G. W. Scupham: Dr. Feinberg's conception of migraine as being essentially an allergic phenomenon is generally accepted. It is analogous to asthma in many respects. Although it is not always an allergic factor that precipitates the episode, that does not alter the pattern. Just as asthma may be precipitated by many exciting factors, entirely foreign to allergic influence, so may migraine. The endocrine aspect deserves more consideration. Just what part the endocrines play in the mechanism of production of headache it is difficult to say.

Dr. Proetz's scheme for study of the subject is splendid. When a patient has sinus infection it does not mean that this is the cause of his headache. The same is true of hypertension.

Robert Von der Heydt.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

EDITORIAL STAFF

DERRICK VAIL, COL. (MC), A.U.S., *Editor-in-Chief* (on active duty)
LAWRENCE T. POST, *Acting Editor-in-Chief*
640 South Kingshighway, Saint Louis 10
WILLIAM H. CRISP, *Consulting Editor*
530 Metropolitan Building, Denver
FREDERICK C. CORDES
384 Post Street, San Francisco
SIR STEWART DUKE-ELDER, BRIG.
63, Harley Street, London
EDWIN B. DUNPHY, LT. COMDR. (M.C.), U.S.N.R.
c/o American Embassy, London
HARRY S. GRADLE
58 East Washington Street, Chicago
S. RODMAN IRVINE, CAPT. (MC), A.U.S. (on active duty)
DONALD J. LYLE
904 Carew Tower, Cincinnati 2
RALPH H. MILLER, CAPT. (MC), A.U.S. (on active duty)
ALGERNON B. REESE
73 East Seventy-first Street, New York
M. URIBE TRONCOSO
500 West End Avenue, New York
F. E. WOODRUFF
824 Metropolitan Building, Saint Louis
ALAN C. WOODS
Wilmer Ophthalmological Institute, Baltimore
EMMA S. BUSS, *Manuscript Editor,* 5428 Delmar Boulevard, Saint Louis 12

Directors: LAWRENCE T. POST, President; WILLIAM L. BENEDICT, Vice-President; DONALD J. LYLE, Secretary and Treasurer; WILLIAM H. CRISP, HARRY S. GRADLE, DERRICK VAIL.

Address original papers, other scientific communications including correspondence, also books for review and reports of society proceedings to *Dr. Lawrence T. Post, 640 South Kingshighway, Saint Louis 10, Missouri.* Manuscripts should be typed in double space, with wide margins.

Exchange copies of medical journals should be sent to *Dr. William H. Crisp, 530 Metropolitan Building, Denver, Colorado.*

Subscriptions, applications for single copies, notices of changes of address, and communications with reference to advertising should be addressed to the *Manager of Subscriptions and Advertising, 837 Carew Tower, Cincinnati 2, Ohio.* Copy of advertisements must be sent to the manager by the fifteenth of the month preceding its appearance.

Author's proofs should be corrected and returned within forty-eight hours to the *Manuscript Editor, Miss Emma S. Buss, 5428 Delmar Boulevard, Saint Louis 12, Missouri. Twenty-five reprints* of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Publishing Company, 450-458 Ahnaip Street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.

REBATES AND THE "AGENCY DISPENSING POLICY"

The Riggs Optical Company has recently announced a new program for the dispensing of glasses to the public. This program is called the Agency Dispensing Policy." While it relates chiefly to business and financial relations between the refractionist and the dispensing optician, certain features are of especial interest to all ophthalmologists in that they apparently give the refractionist the power to regulate somewhat the cost of glasses to the patient. This "Agency Dispensing Policy" as announced is as follows:

1. Dispensing service is available to members of the profession licensed to refract under the laws of the various States in which our Company does business, but only to refractionists practicing within the territory served by our Company.
2. We act as agent for licensed refractionists who are the principals in the dispensing transactions. Occasionally we also act as agent for other dispensing accounts when properly authorized to do so, on the same basis as we act for licensed refractionists.
3. The licensed refractionist appoints us as his agent to dispense for him through the medium of an agency dispensing letter. He determines the amount to be charged and collected for his account by us, and designates the amount either through a signed pricing schedule or by specifying the amount prior to the time of each transaction.
4. Any variations from his schedule must be authorized by the refractionist and a record kept of the authorization.
5. All transactions are charged to the refractionist upon receipt of the prescription or order.
6. All transactions are for cash where credit

is not authorized by the refractionist. Unless credit rating is assured, a sufficient deposit is obtained from the patient at the time the order is placed to cover at least the refractionist's material and service costs, and in every case all unpaid amounts are collected at the time of delivery.

7. Where the refractionist authorizes credit and requests us to do the collecting for him, it is understood that if we do so, acting as his agent, the refractionist bears all credit risks. Statements sent to the patient indicate that transactions are for the account of the refractionist.
8. Agency dispensing accounts are settled monthly. Exceptions to this rule must be requested by the refractionist and agreed to in writing to the refractionist by our Main Office.
9. Casual transients or "drop ins" are not served. Agency dispensing service is not rendered to patients of refractionists who have not properly authorized us to act as their dispensing agents. In no case is dispensing service rendered to any transient patients of a refractionist located outside of our territory of operations.
10. No materials are furnished and no services are rendered unless a prescription or order has been received from the refractionist or record thereof is on file with our Company.
11. All charges to our accounts are based on our current price lists, plus a fixed dispensing fee for the dispensing services.
12. Agency signs describing our dispensing function and procedure are prominently displayed at each of our dispensing offices.
13. Serially numbered receipts are tendered to patients from whom collections are made, which state that the collections are made for the account of the refractionist.
14. The Company reserves the right to decline, through its Main Office, to act as dispensing agent for any refractionist.

August 1943 Riggs Optical Company

The dispensing of glasses to patients concerns the ophthalmologist in two respects: first, his possible participation in the profits resulting from the retail sale, the so-called "rebate system," and second, the price charged the patient for the furnished glasses, which is believed by many ophthalmologists to be excessive.

The participation of the refractionist in the sale of glasses varies between two extremes. On the one hand, is the example of the practitioner in a small community where there is no direct outlet for the sale of glasses to the patient. There the refractionist, after completing his examination, must measure the patient for the frames, determine the type to be worn, order the finished job from the wholesale house, assume the responsibility for the cost thereof, fit the glasses, when received, on the patient, and finally be responsible for alterations and adjustments. Clearly this refractionist, forced by necessity into the position of a retailer, is entitled to the retail profit as a recompense for his services. On the other extreme is the refractionist who, after completing his refraction examination, gives the patient the prescription and directs him to an optical house to have it filled. Thereafter, his task and responsibility are limited to the usual perfunctory examination of the glasses for accuracy and fit, and to the assumption of financial responsibility in case an alteration in the original prescription is necessary, or credit is injudiciously extended to the patient. This latter contingency appears well guarded against in the "Agency Dispensing Policy." As a reward for these minor services and obligations under one guise or another, the refractionist receives back a fat share of the profit ensuing from the sale of the glasses. Disguise this transaction as you will, and distasteful as the term may be both to the refractionist and the optical house, in its last analysis this is nothing more nor less than a straight rebate. While this "Agency Dispensing Policy" is manifestly an effort to deal frankly with this problem, it is open to the criticism that it cloaks a distinctly unsavory transaction, condemned alike by the national ophthalmological societies and by the public, with the mantle of a respectable business procedure.

Certain features of the new policy are,

however, to be commended. First and foremost, it removes the veil of secrecy from the obnoxious business, and provided the patient takes the trouble to inquire into the meaning of the various placards and signs, and into the significance of the receipt given him, he may learn that the refractionist in addition to the fee paid him for professional services, is also receiving a share in the profit from the sale of the glasses. Secondly, and even more important, provided the Riggs Optical Company does not avail itself of the exit offered by "clause 14," the refractionist who does not wish to participate in the retail profits is given a weapon in "clause 3" with which he may strike at the heart of the rebate system and the excessive charge for glasses finally made to his patients.

Under this clause the refractionist is clearly given the power to determine the amount to be charged and collected from the patient for his finished glasses. While no specific limits are set, the obvious minimum would be the wholesale cost of the glasses charged to the refractionist, plus the fixed dispensing fee charged by the Riggs Optical Company (clause 11), plus a small "cushion" charge to provide against loss or subsequent change. Did the refractionist dictate such a price for the patient there could be no rebate of any kind, and the patient could receive his glasses at a considerable reduction over the present prices charged. The obvious maximum would be all the traffic would bear, subject to such ceilings as might be imposed by a government agency. In this case the refractionist would receive the maximum of rebate and the patient, accordingly, charged the maximum for his glasses. Between these two extremes are various possibilities. The refractionist, while refusing to take any rebate, might well feel the patient should properly pay a moderate mark-up on the wholesale price, and any profit finally accruing might be turned over to a charity or some medical institution. Other refractionists, unwilling to give up entirely their profit from the sale of glasses but nevertheless anxious to reduce the final cost to the patient, might compromise the matter in setting the amount to be charged.

What the reaction of the refractionist, ophthalmologist or optometrist, will be to this new policy remains to be seen. If the refractionists avail themselves of clause 3, and set a lower scale of retail prices than those now generally prevailing, what will be the reaction of the Guild opticians who have heretofore enjoyed the former higher scale of prices? Will they be willing to accept the lower scale, and if necessary make a charge for post-sale service to compensate for their reduced income? Or will they bring pressure on the Riggs Optical Company to avail themselves of clause 14, and refuse the business of those who seek a lower-price scale for the patient? What will be the reaction of other wholesale houses to this policy? Will a movement against the present rebate system and for a lower scale of retail prices, if inaugurated in the territory serviced by the Riggs Optical Company, spread to the East and other territories not served by them?

Further developments, if any, will be most interesting to observe.

Alan C. Woods.

SPANISH OPHTHALMOLOGICAL SOCIETY

War, the great destroyer, brings to a standstill many of the finest agencies for progress, both social and scientific. Particularly those countries which must carry on the struggle upon their own soil are so involved in the sheer struggle for existence that they can find few or no op-

portunities for the refinements of civilized life.

The science and art of medicine suffer in common with other agencies for study and research. We in the United States have so far felt the scourge in smaller degree than any other great country concerned. But there is a tendency for important medical societies to suspend their meetings or to reduce the length of those meetings. Essential workers in great institutions have left for service with the armed forces. Medical journals have fewer readers, fewer scientific contributors, and therefore reduced opportunities for service. The exchange of journals and ideas between hostile countries is abolished "for the duration."

Particularly destructive of normal living is the tragic type of warfare which we call "civil war," implying warfare between citizens of the same country. When, in 1936, the forces of Franco opened their campaign against the popularly elected government of Spain, the Sociedad Oftalmologica Hispano-Americana had for thirty-two years played a distinguished part in the development of national and international ophthalmology. Founded in Madrid in 1904, it had held its first eight annual gatherings in that great city; and one of the resolutions adopted at the "Asamblea" held in 1904 in Palma, the capital of the Balearic Islands, declared the intention of holding the next Congress in Madrid, in September, 1936.

In the words of its present secretary, López Enríquez, "Two months before that date, with the labors of organization already well developed, the Glorious National Movement broke out, and it was impossible to meet again until" October 6, 1941.

The inactivity of this important scientific society (and, no doubt, of others equally important in their respective fields) lasted not merely for the period of the civil war, but several years after peace was restored in Spain. The reason for this was apparently a fear on the part of the Falangist government that any large formal gathering, or the activities of any organization not strictly under the supervision of the government, might have mischievous consequences for national stability. The permanent administrative council of the Sociedad Oftalmologica could do nothing beyond guarding the funds of the society. Repeated applications to the government for permission to call a meeting met with refusal. "Considering the international character of the Sociedad, the society's executives were told, and in view of existing circumstances, it was proper to delay the convocation to a later date."

The nineteenth congress of the society was held in Madrid under the presidency of Dr. D. Alejandro Palomar de la Torre. The difficulties of organization encountered by the secretary, Dr. López Enríquez, included an actual lack of information concerning the majority of the members who had been listed before the civil war. The president's short address took notice of the fact that a good many distinguished foreign and national members had died since the gathering of the year 1934. No mention was made officially of some other members who, we may suppose, had gone into voluntary exile from their native land.

To the reader of the society's transactions, the urgent character of the government's refusal to permit an earlier reunion is emphasized by the following quotation from the president's address: "I appreciate that it is a duty of justice . . . to make mention of the spirit of sacrifice, disinterestedness, love of fatherland, and Christian charity, on the part of almost all Spanish ophthalmologists, from the beginning of the Glorious Move-

ment, both those whom it happened to find in the liberated zone, and those who had to carry on, and did it worthily, in the red zone."

Further reference is made to colleagues who, "at the risk of their lives and those of their families, fled from the red zone to take refuge in the liberated zone or placed themselves from the very beginning at the service of the common cause;" and also to the activities of some colleagues who, "having succeeded in escaping from the red zone, and being unable to assemble at an early date in liberated Spain, . . . held high the national banner and professional prestige in foreign countries."

It may too readily be feared that past events have not insured Spain against further fratricidal troubles. But we may rejoice that most of our Spanish colleagues are reunited for scientific activity. The twentieth congress of the Sociedad Hispano-Americana was held in Barcelona in September, 1942. Excellent programs were presented at both annual meetings, and the essays and addresses summarized, together with discussions, are well recorded in the new "Archivos de la Sociedad Oftalmologica Hispano-Americana." This journal, well printed and well edited, appears every month or two. It contains, in addition to the transactions of the annual meetings, a large number of original papers, and some abstracts from the foreign literature.

Among the original papers of the past year and a half are the following: by Leoz Ortín, Ocular phenomena of starvation (already noticed in this Journal); by J. Arjona, Subconjunctival autohemotherapy; by H. Arruga, Corneal transplants from cadaver cornea, and Finding and localizing detachments of the retina; by A. Moreu, Considerations on the ophthalmologic diagnosis of hypophyseal tumors; by A. García Miranda, Avitaminosis in ophthalmology; by M. López Enríquez, Amblyopia of starvation in the alcohol-tobacco group; by J. Sellas Garriga, A case of retinal angiomatosis; by Palomar de la Torre and Palomar Palomar, Concerning the etiology of recurrent juvenile hemorrhages in the vitreous; and by N. Belmonte González, Residual astigmatism in the different ametropias.

W. H. Crisp.

THE LESLIE DANA MEDAL

Last October 17th, the eighteenth award of the Leslie Dana Medal was made. At a well-attended dinner at the Coronado Hotel in Saint Louis, Dr. Walter B. Lancaster became the recipient of this honor. He discussed "The pathetic cases of blindness with good visual acuity." This

Leslie Dana medal.

was an excellent address from the broad-minded viewpoint that we have learned to expect from this man. He is one of those having vision as well as good visual acuity. A man so endowed dares to be a jump ahead of the laboratory, but this is dangerous for those not having critical minds and sound judgment based on long experience.

Father Schwitalla, dean of the Saint Louis University Medical School, presided at the dinner, at which guest speakers were Drs. S. Judd Beach and Everett Goar and Mr. Lewis H. Carris, director emeritus of the National Society for the Prevention of Blindness.

A brief history of this medal may be

of interest. The endowment for the medal was given to the Missouri Association for the Blind by Mr. Leslie Dana in 1925. The trophy itself is a beautiful gold medallion designed by the noted Boston sculptor, Christian Peterson.

The award is to be made annually, based on the following considerations:

a. Long meritorious service for the conservation of vision in prevention and cure of diseases dangerous to the eyesight.

b. Research and instructions in ophthalmology and allied subjects.

c. Social service for the control of eye diseases.

d. Special discoveries in the domain of general science or medicine of exceptional importance in conservation of vision.

The procedure for selecting the winner of the medal is as follows:

The National Society for the Prevention of Blindness will canvass the ophthalmic departments of university medical schools and various other groups interested in prevention of blindness during January and February each year. From the names thus proposed the National Society staff will select a list of not less than three nor more than six persons as nominees for the Dana Medal for the ensuing year. They will also prepare a short biographic sketch for each nominee emphasizing the reason for the selection. As soon as this list of nominees is approved by the Saint Louis Society for the Blind it will be forwarded to the Association for Research in Ophthalmology, Inc., probably before April 1st. The Association for Research in Ophthalmology, Inc., can then send the list to each of its trustees asking them simply to vote one—two—three—and so on, and the person receiving the smallest total vote will receive the medal for the ensuing year. Time and place for the presentation are to be arranged by the Saint Louis Society for the Blind.

Previous recipients of the medal are: Dr. Edward Jackson, Denver, Colorado; Miss Louise Lee Schuyler, New York, New York; Dr. Lucien Howe, Buffalo, New York; Dr. Francis Park Lewis, Buffalo, New York; Dr. Ernst Fuchs, Vienna, Austria; Dr. George Edmund de Schweinitz, Philadelphia, Pennsylvania; Mr. Edward M. Van Cleve, New York, New York; Dr. William Henry Luedde, Saint Louis, Missouri; Dr. Felix de Lapersonne, Paris, France; Dr. William Hamlin Wilder, Chicago, Illinois; Dr. John M. Wheeler, New York, New York; Mrs. Winifred Hathaway, New York, New York; Dr. Ellice M. Alger, New York, New York; Dr. Edward Coleman Ellett, Memphis, Tennessee; Mr. John M. Glenn, New York, New York; Dr. Arnold H. Knapp, New York, New York; Mr. Lewis H. Carris, New York, New York.

The distinguished names on this list indicate the good judgment of those who have selected the recipients and their acceptance the honor in which the award is held.

Awards of this character are stimulating to the profession, and this is properly regarded as one of the most desirable.

Lawrence T. Post.

BOOK NOTICE

THE HUMAN EYE IN ANATOMICAL TRANSPARENCIES. By Peter Kronfield, M.D., Gladys McHugh, and Stephen Polyak, M.D. 99 pages, 34 color paintings. Rochester, N.Y., Bausch and Lomb Optical Company, 1943. Price $6.50.

Here is a volume which comes closer successfully to achieving the goal of a worthy "atlas" of the gross anatomy of eye and orbit than any previous book on the subject. The treatise is divided into three sections. The first consists of a series of 34 colored paintings by Gladys

McHugh. These are paintings of gross anatomic dissections, done on transparent acetate at twice natural size, so that they represent a series of dissections progressing layer by layer through the eye and orbit—in front view and side view.

The accuracy of these pictures is of a high quality, and the artist has succeeded in emphasizing the different important structures in each picture. This characteristic renders the pictures excellent for the student, directly or as an aid to dissection. Each figure is accompanied by a short description of the region shown and of its chief structures.

It is somewhat difficult for the beginner to understand the topographic relations of some of the structures of the eye without the aid of sagittal sections and diagrams—for example, the relationship of orbital septum, orbicularis, levator palpebrae, and Müller's muscle—and, therefore, this volume cannot be a self-sufficient instrument for learning gross ocular anatomy. Likewise, the topographic anatomy of the lacrimal sac and nasolacrimal duct, as well as the anatomy of the zonular region, cannot be grasped readily in layer-by-layer pictures.

However, the second section of the book, an explanatory text by Dr. Peter Kronfeld, aids greatly in the elucidation of some of these more difficult phases. This text contains a systematic description, aided by black-and-white drawings, and then a topographic description, following closely the series of paintings. This portion of the book is clear, concise, and is not overburdened with the mass of mathematical data common to most anatomy texts.

The third section of the book, written by Dr. Stephen Polyak, contains a review of the history of knowledge of ocular anatomy. This makes absorbing reading, but it will undoubtedly be difficult for some readers to transport themselves abruptly from a mass of very material, pictorial facts to the less concrete realm of historical study.

The book is heavily bound with leatherette cover. There is no advertising matter.

Benjamin Milder.

CORRESPONDENCE

On convergence tests

Nov. 13, 1943

Editor, American Journal of Ophthalmology.

I have received several inquiries referring to my article "On convergence tests," in the September, 1943, issue, asking if the angle of convergence could not be calculated, at least approximately, with reference to the Pd and without the use of trigonometric tables. The answer to this is *yes*, it can be found and with a high degree of accuracy by the radian method as follows:

Measure the Pd (interpupillary distance) and the PcB (near point of convergence from spectacle plane—then add 25 mm.). If we call X the angle between the two visual axes at the near point of convergence, then the proportion is Pd is to PcB as angle X is to 57.3 (57.3 degrees is the value of the radian angle). Put in form of an equation $\frac{Pd}{PcB} = \frac{X}{57.3}$. Say the Pd is 62 mm., the PcB is 95 m., then the equation is $\frac{62}{95} = \frac{X}{57.3}$ whence $X = 37.4°$ or 37° 24′. By the sine tables the value comes out 38° 5′. This differs by less than three fourths of one degree from the finding by the radian method. In percentage form the difference is less than 2 percent and is well within the error incident to measurements at very close range.

(Signed) Joseph I. Pascal.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

7

UVEAL TRACT, SYMPATHETIC DISEASE AND AQUEOUS HUMOR

Igersheimer, Joseph. **Intraocular injection of sulfanilamide in a case of purulent iridocyclitis.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1045-1047. (References.)

Marback, H. **A case of congenital aniridia.** Arquivos Brasileiros de Oft., 1942, v. 5, June, pp. 97-101.

The patient, a man aged twenty years, was the only male child of the family. He had five sisters, who were free from the defect. No record of such an anomaly could be obtained for any other member of the family in three generations. The characteristic facies included half-closed eyelids, a horizontal nystagmus, which increased in intensity with increased illumination or upon fixing either distant or near objects. The iris was entirely absent from both eyes. One half of each pupil was clear, the other half was occupied by a semiluxated lens. The right eye presented an opalescent opacity in the lower portion of the cornea, invaded by some conjunctival vessels. The right dislocated lens was entirely opaque, its surface showing vacuoles of varying size. In the lower outer part of this lens there was a 5-mm. pigmented spot made up of deposits which were star-shaped and resembled those of remains of the pupillary membrane. A somewhat similar formation appeared at the seven-o'clock position. With +13.00 sphere each eye obtained vision of 1/6. The tension of each eye was normal. The left cornea had no opacity. The left lens resembled that of the right eye. W. H. Crisp.

Scobee, R. G. **Choroideremia.** Amer. Jour. Ophth., 1943, v. 26, Nov., pp. 1135-1143. (7 figures, references.)

8

GLAUCOMA AND OCULAR TENSION

Ackerman, W. G., and Allen, T. D. **The treatment of hereditary glaucoma.** Illinois Med. Jour., 1942, v. 82, Oct. pp. 295-300.

From the reported genealogical tree

it can be seen that 6 of 14 descendants from the originally affected female ancestor, in three successive generations, had glaucoma. Two of these were male. The average age of all seven affected was 11.6 years, with a span from 7 to 15 years. There was no tendency to hydrophthalmos, and no evidence of alteration of depth of the anterior chamber, or of the presence of an enlarged lens. Myopia was found in two cases. The glaucoma was of the simple chronic type. As to treatment, intraocular tension responded poorly if at all to miotics alone. As to surgical procedure iridectomy and posterior sclerotomy were found ineffective. In one patient one goniotomy has been effective for the last 29 months in one eye, whereas in the other eye five goniotomies, two cyclodialyses, and a deep root iridectomy were without result, but one iridencleisis has since controlled the condition for 19 months. Two goniotomies performed a year apart on each eye of another member of the family have apparently sufficed for nine months to date. In another, one goniotomy on the right eye, performed 18 months ago, and two on the left eye, performed 18 and 6 months ago, have controlled the condition to date. In another member a single goniotomy of the left eye has controlled tension, fields, and vision for eight months to date. The technique of goniotomy and the aftercare are described in detail. The authors add that miotics are of value in maintaining lowered tension before and after surgery. Various types of visual defect are reported. Melchiore Lombardo.

Gibson, G. G. **Transscleral lacrimal canaliculus transplants.** Trans. Amer. Ophth. Soc., 1942, v. 40, pp. 499-515.

Sixteen experimental transplants of a portion of the lacrimal canaliculus into the sclera of dog eyes are described in detail. This procedure was developed by the author with the immediate objective of investigating the various early steps in the establishment of an epithelial tube through the sclera with the hope of finding a type of filtration operation that might be added to the operative armamentarium in the control of glaucoma.

A very cautious evaluation of the procedure is presented. After due consideration of possible objections and pitfalls, the operation was tried on a patient with advanced glaucoma who had very little vision to lose. Marked reduction in intraocular tension was produced during the first three weeks. Later the external end of the canaliculus became closed by subconjunctival scar tissue and the tension returned to its preoperative level.

The author emphasizes that the work is still in the experimental stage, but he seems satisfied of the feasibility of the procedure after certain technical improvements are made and there has been further clinical trial. (5 illustrations.) David O. Harrington.

Knighton, W. S. **Reoperations for glaucoma.** Arch. of Ophth., 1943, v. 30, Oct., pp. 499-504.

The return of hypertension after a glaucoma operation is of fairly frequent occurrence. In such cases, the surgeon has already selected and performed the operation that he considered the most likely to succeed. The failure of the operation to control pressure leaves him with a considerable handicap. After the operation the eye is in a less favorable state because of fibrosis, mechanical difficulties, physiologic changes and other, unknown, factors. Filtration operations that have failed

may occasionally be made to work by removing plugs of uveal or scleral tissue from the fistula. Failures in filtration that result from cicatrization of the conjunctiva do not respond to attempts to reëstablish filtration by again dissecting the flap. Very careful study of the eye, including gonioscopic observation, is always indicated before reoperation is attempted.

John C. Long.

9

CRYSTALLINE LENS

Burch, E. P. **Safeguards in cataract surgery.** Jour. Florida Med. Assoc., 1943, v. 29, Jan., p. 307.

Burch describes his procedures for avoiding complications in cataract surgery. These include methods of anesthesia and akinesia, and the use of corneoscleral sutures with conjunctival apron for covering the section. Retrobulbar anesthesia is advised.

T. M. Shapira.

Courtney, R. H. **Endophthalmitis with secondary glaucoma accompanying absorption of the crystalline lens.** Trans. Amer. Ophth. Soc., 1942, v. 40, pp. 355-369.

Seven cases are reported which suggest a hitherto unrecognized clinical entity: namely, a late endophthalmitis in one eye due to sensitivity to lens protein, the latter having resulted from an operation on the fellow eye some years previously. The author believes that spontaneous rupture of the lens capsule, found clinically in one case and histologically in another, is the causative factor. The patients showed hypersensitivity to lens protein and in two instances there was evidence of spontaneous rupture of the lens capsule. The cases reported exhibited the following clinical picture: (1) marked ciliary injection; (2) heavy keratic precipitates of the epithelioid type, the precipitates occasionally being so numerous and so large as to coalesce and obscure any view of the interior of the eye; (3) markedly increased intraocular tension; (4) greatly increased sensitivity to lens protein; (5) unmistakable evidence of spontaneous rupture of the lens capsule. When it is clear that the endophthalmitis results from absorption of the lens cortex, the correct treatment is removal of the lens matter by operation, an intracapsular extraction being highly desirable. Failing this, the use of thorough irrigation of the anterior chamber is urged. In the author's cases, after removal of the lens from the eye all the foregoing signs of endophthalmitis and secondary glaucoma disappeared by the time of the first dressing, and convalescence was uneventful. (5 illustrations, references.) David O. Harrington.

Moehle, Walter. **The use of conjunctival flaps in cataract extraction.** Trans. Amer. Ophth. Soc., 1942, v. 40, pp. 527-546.

After a fairly comprehensive review of the history and literature of conjunctival flaps in cataract surgery, the author presents his own technique of extraction of the lens through a vertical conjunctival slit. He feels that this method embodies certain distinct advantages as follows: (1) The conjunctiva alone is undermined. (2) Unnecessary conjunctival bleeding is avoided by the direction of incision. (3) Keratome incision into the anterior chamber and enlargement of the incision with scissors is easily accomplished. (4) Suture control of the wound lips is easy. (5) Iris and lens capsule are easily accessible. (6) Eversion of the

cornea is impossible. (7) Prolapse of the iris is diminished in frequency. (8) There is less danger of vitreous loss. (9) Healing is accelerated and postoperative astigmatism is reduced.

All extractions were through keratome incision enlarged by scissors, with full iridectomy and extracapsular extraction. (One illustration, extensive statistical tables, bibliography.)

David O. Harrington.

10

RETINA AND VITREOUS

Campos, Èvaldo. **Neuroretinitis with macular star.** Arquivos Brasileiros de Oft., 1942, v. 5, June, pp. 131-136.

This anomaly was observed in a boy of eleven years. The vision of the right eye was reduced to light perception, without light projection. The vision of the left eye was 1.5. From the fovea, spindle-shaped white lines ran out at perfectly regular intervals, like the spokes of a wheel. The total area involved was larger than the disc, including some edema. The disc was decidedly edematous, the vessels distended, especially the veins; and there were small hemorrhagic foci in the area of edema. The urine contained only slight traces of albumen and was otherwise practically normal. The author excludes a renal origin. There were physical stigmata of syphilis, and the author is disposed to attribute the ocular condition to this disease. (11 references.)

W. H. Crisp.

Cross, A. G. **Angioma of the retina.** Brit. Jour. Ophth., 1943, v. 17, Aug., pp. 372-373. (See Section 15, Tumors.)

Gaynon, I. E., and Asbury, M. K. **Ocular findings in a case of periarteritis nodosa.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1072-1076. (References.)

Kauffman, M. L. **Lipemia retinalis.** Amer. Jour. Ophth., 1943, v. 26, Nov., pp. 1205-1208. (References.)

Marburg, Otto. **Inclusion bodies and late fate of ganglion cells in infantile amaurotic family idiocy.** Arch. of Neurology and Psychiatry, 1943, v. 49, May, p. 708. (See Section 17, Systemic diseases and parasites.)

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Barrenechea, S., Contardo, R., Arentsen, J., and Jasmen, A. **Treatment of optic neuritis, retrobulbar or not, with nicotinic acid.** Oftalmologia Ibero Americana, 1943, v. 5, no. 1, pp. 7-21 (in Spanish) and 21-29 (in English).

The author treated five cases of optic neuritis with nicotinic acid. Four of the cases were retrobulbar. The dose used was 300 mg. daily. There was rapid improvement in central vision, visual fields, perception of colors, and subjective symptoms, within the first ten days of treatment. Each case is described in detail.

12

VISUAL TRACTS AND CENTERS

Fralick, F. B. **The treatment of amblyopia.** Amer. Jour. Ophth., 1943, v. 26, Nov., pp. 1195-1197.

Hughes, E. B. C. **Injury to the opticochiasmal junction.** Brit. Jour. Ophth., 1943, v. 27, Aug., pp. 367-371.

In the case here reported there was injury in the left frontal region with a fracture of the left frontal bone, traversing the left frontal sinus, running along the floor of the left anterior fossa lateral to the optic canal, and then passing down into the left middle

fossa. The course of the fracture was verified in two operative procedures. No definite fracture into the left optic canal was demonstrated and no fracture of the left anterior clinoid process was demonstrable by X ray or at the time of operation. Two days after injury, a left epidural hematoma was exposed and removed through a temporal craniotomy. Postoperatively, there was severe loss of vision in the left eye and a partial third-nerve paresis. The right visual field was normal. The left visual field showed doubtful perception of light in the upper nasal field only.

A week after the operation, the patient had a clinical attack of meningitis, which lasted only a few days and responded rapidly to sulfapyridine. At this time the third-nerve paresis began to progress. It became complete thirteen days after injury. Visual acuity in the left eye had improved by this time to good counting of fingers in the upper nasal field only. The right field was passed as normal again. During the next few weeks the patient remained mentally confused, and a left frontal intracerebral aërocele was suspected and confirmed by X ray. It was not until ten weeks after the injury that the patient's condition was good enough to warrant full quantitative perimetry. A large island of vision in the upper nasal field was found on the left side, together with a little macular vision. Acuity was 1/60. In the right field, however, a large upper temporal quadrantic defect was found together with some depression in the lower temporal field. This field pattern remained stationary until the second operation, sixteen weeks after injury.

At operation, the posterior part of the anterior fossa was found to be the seat of massive adhesions extending back as far as the sphenoid ridge and enveloping the intracranial part of the left optic nerve as far as the chiasmal junction. The chiasm itself appeared to be normal. The nerve was freed from its adhesions and the operation was terminated by drainage of the frontal cyst resulting from the aërocele.

There was a marked improvement in the patient's mental state postoperatively, and an equally startling and unexpected change took place in his visual fields. Vision in the left eye rapidly improved within fourteen days from 1/60 to 6/36. Perimetry showed this to be due to a general enlargement of the island of vision. The field in the right eye also improved until only a moderate-sized upper temporal scotoma remained.

The site of this patient's injury must have been either in the termination of the left nerve or in the anterior chiasmal angle. The lesion affected mainly the fibers of the left nerve but also caught the knee of crossed fibers from the lower nasal retina of the right eye. (Fields, references.)

Edna M. Reynolds.

Long, A. E. **Amaurosis following nasal hemorrhage.** Amer. Jour. Ophth., 1943, v. 26, Nov., pp. 1179-1182. (References.)

Verhoeff, F. H. **A new answer to the question of macular sparing.** Arch. of Ophth., 1943, v. 30, Oct., pp. 421-425.

The destruction of one optic tract results in splitting of the macula as detected by field tests. Apparent sparing of the macula occurs after complete destruction of one occipital lobe. This apparent sparing is explained by loss of integration between the seeing field and the blind field in the conscious visual cortical area in use. This loss results in the employment of an eccentric

retinal point for fixation and slipping of fixation occurs easily. The true macular field is actually split but field tests show it to be more or less spared.

Lesions of the optic tract do not disturb the integration between the seeing field and the blind field. In these lesions there is no eccentric fixation and splitting of the macula is therefore found in field tests. Even after complete destruction of one occipital lobe, macular splitting may be found if integration in the conscious visual area in use is not too greatly disturbed. The author states that this theory needs additional facts before it can be unreservedly accepted. John C. Long.

13

EYEBALL AND ORBIT

Courtney, R. H. **Endophthalmitis with secondary glaucoma accompanying absorption of the crystalline lens.** Trans. Amer. Ophth. Soc., 1942, v. 40, pp. 355-369. (See Section 9, Crystalline lens.)

Dunnington, J. H. and Berke, R. N. **Exophthalmos due to chronic orbital myositis.** Arch. of Ophth., 1943, v. 30, Oct., pp. 446-465.

Chronic orbital myositis has at times been described under the less desirable names of idiopathic myositis, Zeker's waxy degeneration, pseudotumor, and exophthalmic ophthalmoplegia. This condition is a nonspecific, chronic inflammatory disease of the extraocular muscles of unknown causation. Microscopic examination of the involved muscle shows lymphocytic infiltration, fibrosis, and degeneration of muscle fibers. The muscles are enlarged, pale, and cartilaginous. The disease is relatively painless unless keratitis results from exposure. There is edema of the lids, moderate to severe exophthalmos, and marked diplopia. There is distinct limitation of motion of the eyeball, especially upward, and inelasticity of the affected muscles. The limitation of motion is best demonstrated by grasping the anesthetized eye with forceps. Strong resistance is encountered when an attempt is made to stretch the involved muscles.

There is no recognized form of treatment for chronic orbital myositis. Surgery is to be avoided if the correct diagnosis can be made, as the condition is practically always rendered worse by surgery. Radiation is not of value. The prognosis for spontaneous recovery is good if surgery is not undertaken. Treatment should be directed toward preservation of the cornea.

The authors report four cases of chronic orbital myositis. All of these patients were operated upon. A detailed pathological report on the tissues is given. All but one of the patients showed severe permanent limitation of motion. (Bibliography.)

John C. Long.

Snow, W. J. **Orbital complications in suppurative sinus disease.** Texas State Jour. Med., 1943, v. 38, Jan., p. 557.

The author describes five cases with swollen lids, conjunctival edema, proptosis, displacement and fixation of the globe, and a history of recent onset. These cases were not treated uniformly, but the author is convinced that early surgery is desirable. He believes that the external ethmofrontal approach affords good exposure and good drainage with a minimum of surgery. He is also of the opinion that classical operations on the sinuses should be avoided during the fulminating stage of these infections. T. M. Shapira.

Verhoeff, F. H. **Improved technique for implantation of a ball in Tenon's capsule.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1057-1061. (References.)

14

EYELIDS AND LACRIMAL APPARATUS

Ellis, O. H. **A combined ptosis operation.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1048-1052; also Trans. Pacific Coast Oto-Ophth. Soc., 1942, 30th mtg. (5 drawings, references.)

Paula Xavier, J. de. **Blepharitis and pyoctanin.** Rev. Med. do Paraná, 1943, Jan., abstracted in Arquivos Brasileiros de Oft., 1943, v. 6, April, pp. 39-42.

The generic denomination of pyoctanin includes various substances derived from anilin, endowed with cicatrizing and antiseptic qualities, without being toxic or irritating. Examples are methylene violet, auramin, methylene green, and methylene blue. The drug is used in the form of a pencil mounted in a small wooden case. Van Lint and others have advised cleaning the ciliary margin with benzine in order to remove all greasiness, then passing the pencil, moistened in 40-percent alcohol, along the roots of the lashes. The application is made every second day. The author's cases showed no example of intolerance. Any objectionable staining of the lids can be disguised by the use of tinted glasses. Failures are attributed to inadequate care as to cleansing of the lid margins. (Abstract in Portuguese by W. Belfort Mattos.)

Potter, W. B. **Bilateral congenital coloboma of upper lids.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1087-1089. (One illustration, references.)

15

TUMORS

Cross, A. G. **Angioma of the retina.** Brit. Jour. Ophth., 1943, v. 27, Aug., pp. 372-373.

In the case of angiomatosis retinae now reported there was such rapid increase in size within a week's time that a diagnosis of malignant growth was made and the eye was enucleated. It is probable that if the eye had not been removed retinal gliosis would have occurred, followed by complete degeneration of the eye. This case confirms the belief that angiomatous growth is the initial abnormality in cases of Hippel's disease. (One illustration, references.)

Edna M. Reynolds.

Ray, B. S., and McLean, J. M. **Combined intracranial and orbital operation for retinoblastoma.** Arch. of Ophth., 1943, v. 30, Oct., pp. 437-445.

Extension of retinoblastoma into the optic nerve is a very common occurrence. It is believed that the standard operation of enucleation is inadequate in nearly 50 percent of cases of retinoblastoma. Exenteration of the orbit or secondary intracranial resection of the nerve is unsatisfactory. Irradiation of the stump of the optic nerve after enucleation has improved results but is not wholly satisfactory.

As the most complete method of removing the tumor, the authors propose intracranial resection of the optic nerve followed by enucleation. The intracranial operation is performed through an incision like that routinely employed by the neurosurgeons in the "hypophyseal approach". The nerve is cut at the chiasm and at the optic foramen and is removed. The optic foramen is coagulated by the electrosurgical unit and then plugged with a piece of temporal muscle. Twelve to thirteen days later the eye is enucleated. The remainder of the optic nerve is removed by traction. The intracranial operation is relatively safe and is well tolerated even by infants.

It is safer to do the intracranial operation before enucleation, because one is then more certain of removing all of

the nerve and there is less danger of intracranial infection. The chief disadvantage is that in approximately 50 percent of the cases the intracranial operation is unnecessary, as enucleation shows that the nerve was not involved. The authors report the cases of two patients treated by the two-stage operation. Both patients stood the surgery very well.

John C. Long.

16

INJURIES

Alvis, E. O. **Wartime-patient phase of industrial ophthalmology.** Jour. Indiana State Med. Assoc., 1943, v. 36, May, p. 235.

Though statistics on the comparative increase of eye injuries since war production has been speeded up are not available, the author from his own experience feels that there has been an increase in the number of industrial eye injuries. Their number can be reduced by proper education, coöperation between employer and employee, and ample provision for making a plant physically safe. Guards, goggles, shields, and other protective devices are absolutely essential. Fluorescent lighting is considered unfavorable from a safety standpoint.

Injuries from emery, tool particles, chemicals, and actinic rays are discussed and suggestions given. Early antiseptic care of eye injuries is highly stressed. The employee is warned against the self-appointed "foreign body remover," usually a fellow employee with no regard for asepsis and whose manipulations at times end disastrously. Francis M. Crage.

Beil, W. C. **Closure of the sclera after removal of intraocular foreign body by the posterior route.** Amer. Jour. Ophth., 1943, v. 26, Nov., pp. 1210-1211.

Belfort Mattos, W. **A cilium twenty years in the anterior chamber.** Arquivos Brasileiros de Oft., 1943, v. 6, April, pp. 38-39.

The patient, a man of 28 years, had received an injury to the right eye at the age of eight years. There was a small central leucoma of the cornea. From the center of the leucoma a filament, not unlike those seen in remains of the pupillary membrane, proceeded toward the iris. With movements of the iris, the filament became displaced on the anterior surface of that structure. There was no pericorneal injection or other anomaly. It was found impossible to seize the filament through a small opening at the edge of the cornea, but expulsion was obtained by somewhat forcible lavage. (One illustration.)

W. H. Crisp.

Clark, W. B. **Procedures and appliances that are helpful in treating industrial ocular injuries.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1054-1057. (2 illustrations.)

Cordes, F. C. **Nonsurgical aspect of ocular war injuries.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1062-1071. (References.)

Kilgore, G. L. **An experimental study of iridodialysis.** Trans. Amer. Ophth. Soc., 1942, v. 40, pp. 516-526.

An experimental study of the mechanism of production, the early and late clinical picture, and the prognosis of iridodialysis produced by trauma is presented.

The author concludes that iridodialysis may be produced by sudden blows on the corneal surface at or anterior to the corneoscleral limbus. The iris is

not readily torn by contusions wherein the force radiates slowly from the point of injury. Considerable damage to the intraocular tissues is associated with traumatic iridodialysis. The intraocular pathology of iridodialysis cannot be determined by the uusual methods of eye examination, and a guarded prognosis should be given in such cases. (16 illustrations, references.)

David O. Harrington.

Kolen, A. A. **Soviet military ophthalmology.** Lancet, 1943, v. 244, June 26, p. 804.

When war broke out, base hospitals throughout the Soviet Union were equipped with X-ray apparatus and giant electromagnets. Ophthalmologists were grouped with otolaryngologists and stomatologists in order to combine the treatment of face wounds in general. The ophthalmic service was so efficient that large numbers of the ocular casualties were speedily returned to the ranks and the labor front.

For years past the clinic at Novosibirsk has paid especial attention to plastic operations. Several highly satisfactory procedures are described by Kolen, including reconstruction of the conjunctival sac without grafting of new tissue, but by stretching shrivelled surfaces, adapting a prothesis, and allowing the conjunctival tissue to proliferate so as eventually to cover the whole affected area without secondary shrinkage.

In wounds involving the orbital borders, scars can be separated, dissected out, and the gap filled with a pedicled flap taken from the adjacent subcutaneous cellular tissue. Such flaps readily became engrafted, and, without shrinking, afford excellent cosmetic results. Defects of mucous membrane of the lids, with adhesions, are repaired by what the author calls the "transplantation flap-method" which consists of implanting upon the raw surfaces strips of mucous membrane from the lips. In reconstructing eyelids a "flap-pocket" is employed, consisting in the planting of strips of lip membrane in a pocket beneath healthy adjacent skin and, later, intercalating a slice of ear cartilage between the skin and the membrane; as a final step, all components (skin, cartilage, and mucous membrane) are transplanted by pedicle, giving in effect a ready-made lid.

Whereas surgeons have commonly deemed it wise to wait until shrinking was complete, a process frequently consuming months or years, in wartime such delays cannot be allowed. Moscow surgeons operate soon after the receipt of injuries, especially when uncomplicated by fracture or extensive suppuration; and they obtain good results even as early as two to four months after injuries are received.

Contracted scars in the temporal region adhering to the bone are likely to involve the meninges. For these and those invading the deep nasal area, where fistula or other complications might follow, special care is taken in all transplanting and the adjusting of pedicled flaps.

At the Novosibirsk clinic segments of the ciliary body have been excised, and the author cites an instance in which a ciliary sarcoma was removed without loss of sight to the affected eye. War-time successes in the same field suggest the foundation of a new operative sphere in the eye—surgery of the ciliary body. Burton Chance.

17

SYSTEMIC DISEASES AND PARASITES

Abreu Fialho, Sylvio. **Cysticercus cellulosae in the anterior chamber.** Ar-

quivos Brasileiros de Oft., 1942, v. 5, June, p. 142. (Abstract from O Hospital, 1942, v. 21, no. 5, May.)

The original article related a case of cysticercus in the anterior chamber, with references to the national medical literature of Brazil, which records only three cases. In the case now reported, the cysticercus was in the inner part of the angle of the anterior chamber, with adhesions to the iris. Operation consisted of simple incision at the limbus above. The diagnosis was confirmed by laboratory examination. The larva was dead and the cyst calcified. The eye retained vision of 1/10.

(Abstract in Portuguese by Alfredo Rocco.)

Constantine, E. F. **Tuberous sclerosis.** Arch. of Ophth., 1943, v. 30, Oct., pp. 494-498.

Tuberous sclerosis is a clinical entity characterized by adenoma sebaceum, convulsions, mental deficiency, and fundus lesions. The author observed this condition in a ten-year-old boy. The boy had been normal until six months of age at which time generalized twitching developed and it was noticed that he was cross-eyed. He had had several attacks diagnosed as encephalitis, and numerous generalized convulsions. He did very poorly in school and was not coöperative. On examination typical adenoma sebaceum of the face was found. There was a convergent strabismus. The vision of the right eye was 20/20−2 and of the left eye 20/400 eccentrically. The right fundus showed a large flat whitish area below the disc and a smaller similar area above the disc in which there were a few cysts. The left fundus showed three tremendous cystic swellings, one on the disc and one above and below the macula. Roentgenograms of the skull showed several calcified areas, probably in the choroidal plexus. An electroëncephalogram was suggestive of a neoplasm. John C. Long.

Edgerton, A. E. **Herpes zoster ophthalmicus.** Trans. Amer. Ophth. Soc., 1942, v. 40, pp. 390-439.

This exhaustive treatise on the subject of herpes zoster ophthalmicus reviews the literature of the subject, including the history of the disease. The present status of the subject is outlined under the following headings: anatomy, age, sex, seasonal occurrence, exposure, prodromal symptoms, pain, vesicles, lymphatic glands, eyelids, conjunctiva, ocular complications, corneal anesthesia, cornea, intraocular tension, iris, sclera, nasal nerve, lacrimal nerve, facial nerve, sympathetic disturbances, Argyll Robertson pupil, sympathetic ophthalmia, exophthalmos, muscle paralysis, third nerve, third, fourth, and sixth nerves, bilateral cases, recurrent cases, optic-nerve involvement, other nerve involvement, types (epidemic and symptomatic), chicken pox, herpes simplex, pathology, optic nerve, retina and choroid, glaucoma, adjoining ganglia, spinal fluid and blood, prognosis, diagnosis, treatment, and conclusions.

The author reports his own cases and his experiences in diagnosis and treatment. In conclusion he presents a statistical analysis of the composite clinical and pathologic picture as gleaned from the literature. (Extensive bibliography.) David O. Harrington.

Magalhães, Octavio de. **Scorpionism and visual apparatus.** Arquivos Brasileiros de Oft., 1942, v. 5, June, pp. 141-142. (Abstract from O Hospital, 1942, v. 21, no. 5, May.)

The article is a survey of the action of this poison upon animals and man,

from the point of view of the eye. In mild cases there are no changes in the visual apparatus, but in severe or medium cases the ocular changes are marked. They may include changes in neuromotor control, inducing nystagmus, convergent strabismus, palpebral ptosis, conjugate deviation of the eyes and the head, and disturbances of the intrinsic musculature of the eye in the form of mydriasis or miosis; sensory disturbances in the form of blindness or diminution of vision; and finally disturbances of the vascular and secretory apparatus of the eye, with lacrimation, bluism coloring of the sclera, and retinal hemorrhages.

Animal experiments are recorded. The author also reports a case of poisoning in a child of two years who had been stung by a scorpion. In this case, in spite of every attempt at treatment, including antiscorpionic serum, the child developed a generalized hypersecretion, accompained later by blue coloring of the scleras and terminating in steady deterioration of the general condition, with acute pulmonary edema and death. The original article is said to contain a fairly abundant bibliography. (Abstract in Portuguese by Alfredo Rocco.)

Marburg, Otto. **Inclusion bodies and late fate of ganglion cells in infantile amaurotic family idiocy.** Arch. of Neurology and Psychiatry, 1943, v. 49, May, p. 708.

The author investigated the pathologic changes in infantile amaurotic family idiocy. He discovered the existence of inclusion bodies as well as severe secondary cell changes. The inclusion bodies are partly argentophilic and partly argentophobic, depending on the kind of fat which forms their basis. They are usually associated with convulsive states, or myoclonia. According to their origin the inclusion bodies may be differentiated into three types. One type is derived from fat, another type from changes in the axons and fibrils, and the third type is formed by nuclear excretion. Only the first two types are associated with amaurotic family idiocy, and only they can be considered as evidence of degenerative metabolic processes, while the third type is seen in infectious diseases.

Every ganglion cell has a functioning part, the fibrils, and a vegetative part, the cell plasma. In amaurotic family idiocy the fibrils remain for a long time intact, and the severe changes in the plasma may produce few clinical signs and symptoms. Only in diseases of longer duration, when the fibrils, too, become deranged, do we find corresponding severe clinical signs. The atonic asthenia is due to cholinergic and adrenergic factors, obviously the result of changes in the thymus and the adrenals. It is similar to the asthenia found in myasthenia and in Addison's disease. R. Grunfeld.

Paulo, A., Jr. **Ophthalmopathies of tuberculous nature.** Arquivos Brasileiros de Oft., 1942, v. 5, June, pp. 101-117.

This is an inaugural address delivered in the Escola Paulista de Medicina. It reviews the general subject of ocular tuberculosis, with numerous references to the literature, and with special discussion of phlyctenulosis, hemorrhage into the vitreous, and the disease known as Boeck's sarcoid. Of the last condition the author cites an individual case, in a woman of 45 years. There were deep orbital pains on the right side, which were said to have existed since sudden loss of vision some time previously. The left eye had tem-

porary obcurations of vision, and showed a 50-percent loss of visual acuity. There were nodules in the eyelids, in the frontal region, in the ala of the nose, and in the right malar region. After a month of treatment with tuberculin, all the subcutaneous nodules underwent absorption. In the course of a year glaucoma developed in each eye, and antiglaucomatous sclerectomies were performed. The right eye retained its vision, but the left eye became entirely blind. W. H. Crisp.

18

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Belfort Mattos, W. **Need for medical syndicalism among the oculists.** Arquivos Brasileiros de Oft., 1942, v. 5, June, pp. 137-141.

This address, delivered upon taking over the presidency of the Ophthalmological Society of São Paulo, Brazil, talks very frankly of the inadequacy of the economic status of ophthalmology in Brazil. In that country there are not more than 800 ophthalmologists for a population of about 40 million. Although the Brazilian law requires syndicalization (a sort of trade union development) of physicians, the ophthalmologists are said to make little use of this means of coördinated effort. The author complains that, in the very proper desire of obtaining a good clientele and good economic standing, the new oculist, like other young physicians, seeks the poorly remunerative work of so-called benefit societies, or of insurance companies, or sacrifices all his mornings to the advantage of those exploiters of medical services, the charity clinics. The young oculist argues that he is there to learn and practice, forgetting that in all professions the apprentice also is paid for his work. Frequently he works for an older colleague. He begins to run hither and thither in an automobile bought on "easy payments", to appear in medical gatherings and take part in discussions and dinners, and competes for the post of junior helper to the junior assistant in a charitable institution, in order to be able to use that position as an advertisement at the head of his prescription blanks, which he gets printed by some drug concern. The oculist neglects the smaller communities, preferring to remain in the large cities.

These and many other things Belfort Mattos says in order to demonstrate that within the Ophthalmological Society of São Paulo, side by side with the study and development of Brazilian ophthalmology, ophthalmologists can and should strive for a better economic and social condition of their specialty. W. H. Crisp.

Chance, Burton. **A survey of the state of ophthalmology in Philadelphia at the time of the founding of the Section on Ophthalmology at the College of Physicians.** Amer. Jour. Ophth., 1943, v. 26, Nov., pp. 1164-1170.

De Brito Conde, Herminio. **Inaugural address of the course on ocular hygiene.** Arquivos Brasileiros de Oft., 1942, v. 5, June, pp. 117-130.

The author, who is General Secretary of the Brazilian National League for the Prevention of Blindness, delivered the opening address in a series organized by the Brazilian General Secretariat of Health and Assistance. He insists on the need for an ocular conscience, cites from the literature classical examples of the influence of visual disturbance upon the career of the individual (especially with regard to an author named Machado de Assis), and

supports the belief that continuous near work may induce or aggravate pathological ocular conditions.

W. H. Crisp.

Lowenfeld, Berhold. **Partially seeing children in schools for the blind.** Outlook for the Blind, 1942, v. 36, Dec., p. 287.

This article is a survey of the admission requirements of partially seeing children for admission into schools for the blind. Legal requirements with regard to amount of vision are given and also the amounts of vision actually recognized for admission into various schools. Questionnaires returned by the schools furnished the information.

In most states a definite specification in regard to the amount of vision is not included in the legal requirements for admission. In 88 percent of the schools it is found impossible to adhere to a strict definition of blindness in dealing with admissions. The author explains the factors which cause requirements to vary. Francis M. Crage.

Norris, M. A. **Review of cases admitted on the eye service at the Riley Hospital from 1936 to 1942.** Jour. Indiana State Med. Assoc., 1942, v. 36, May, p. 239.

The cases admitted to the hospital during this six-year period included 114 congenital anomalies, 54 injuries, five retinal detachments, 126 inflammatory conditions, 15 tumors, and 212 cases of strabismus. In the reviews are noted the important findings, ideal aims, and the general treatment employed in some of the cases.

Francis M. Crage.

Oak, Lura. **The Massachusetts vision test, an improved method for school vision testing.** Amer. Jour. Public Health etc., 1942, v. 32, Oct., pp. 1105-1109. See Section 1, General methods of diagnosis.

Sloane, A. E., and Gallagher, J. R. **A summary of findings at the eye examination of preparatory-school boys.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1076-1083. (4 tables, references.)

Woods, A. C. **The story of the Red Cross Institute for the Blind (1918-1925) in relation to the present problem of the war-blinded.** Amer. Jour. Ophth., 1943, v. 26, Oct., pp. 1011-1024. (References.)

19

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Browman, L. G., and Ramsey, F. **Embryology of microphthalmos in rattus norvegicus.** Arch. of Ophth., 1943, v. 30, Sept., pp. 338-351.

There are a number of influences which affect and arrest the eye in its embryonic growth: for example, mechanical injuries, or abnormal pressures; infections, toxicoses, vitamin or other nutritional deficiencies, metabolic and endocrine disturbances in the mother ante partum, and interference with the blood supply to the embryo or to the developing primordia. Certain abnormalities of the eye have been shown to be inherited.

In order to study the heredity, the authors started with a female rat with bilateral microphthalmos and mated her with a normal-eyed litter mate; one microphthalmic animal appeared. Brother-sister matings of this strain were continued in an attempt to increase the proportion of small-eyed animals.

The average percentage of microphthalmic rats increased from a low of 19

to over 85 in the seventh generation. It is of interest to note that no litter in which all animals had some degree of microphthalmos was observed until the seventh generation. In this generation 7 of 20 females produced litters in which all of the young exhibited some degree of microphthalmos. In the present investigation 22 sixth-generation microphthalmic females were used to obtain 51 embryos and fetuses.

The development of the normal eye in a series of embryos from the microphthalmic females was followed from the optic-vesicle stage to the stage just before delivery. A table of this development is given. Only two brain anomalies associated with microphthalmos were observed. Autopsy disclosed that the optic nerves were missing in 19 of 48 adult rats with apparently normal-sized eyes, and with no opacities or other abnormalities. The peculiar association of absence of the hyaloid artery with a well-developed annular blood supply probably accounts for the appearance of a normal eyeball without an optic nerve.

In none of the 10.5-to-12-day-old embryos studied did the optic vesicle and the optic cup reveal abnormalities. It would therefore seem that the critical period in the formation of microphthalmos in this strain of rats is at the time of formation of the embryonic blood supply to the eye, namely, at 12 to 13 days. Other structures in the eye and orbit were also involved. (8 figures, 35 references.)

Ralph W. Danielson.

Gregory, P. W., Mead, S. W., and Regan, W. M. **A congenital hereditary eye defect of cattle.** Jour. of Heredity, 1943, v. 34, April, p. 125.

Inbreeding experiments with Jersey cows, designed to fix the factors for high milk and butterfat production, have brought out a recessive change in the form of a congenital type of cataract. This was found in pure-bred Jerseys at the University of California. The lenses were smaller than normal and were dislocated. Ophthalmologic studies made included microscopic sectioning of affected eyes.

Francis M. Crage.

Sverdlick, José. **Observations on the structure of the retinal cells.** Arch. de Histologia Normal y Patological, 1943, v. 1, March, 13 pp. (See Section 10, Retina and vitreous.)

PAN-AMERICAN NOTES

Edited by Dr. M. Uribe Troncoso
500 West End Avenue, New York

Communications should reach the Editor by the twelfth of the month

Miscellaneous

Argentina. The Argentine Committee for the Prevention of Blindness and Trachoma has appointed the following physicians to the Board of Directors for 1943-1944: Dr. J. Lijo Pavía, president; Pio Pandolfo, first vice-president; Enriquo Monchet, second vice-president; Carlos R. Porto, treasurer; Dudley S. Drabble and Buido S. Longoni, secretaries.

The Argentine Ophthalmological Society held a theoretical-practical course on strabismus under the supervision of Dr. Jorge Malbran. The course consisted of a series of five lectures held at the Argentine Medical Association and three surgical demonstrations held in the Santa Lucia Ophthalmic Hospital. The subjects included in the course were: Mechanics of ocular movements; Binocular vision; Fusion; Disturbances of muscular equilibrium; Latent strabismus or heterophorias; Vision of the patient with strabismus; Etiopathogeny of strabismus; Convergent and divergent strabismus; Oblique strabismus; Treatment of strabismus; Paralyses of the extrinsic muscles of the eye; Paralyses of the third pair of supranuclear paralyses.

Brazil. The Ophthalmic Board of Examination in Brazil (Consellio Nacional de Oftalmologia) has awarded certificates of proficiency to nearly 150 physicians in all the territory of Brazil.

Permanent postgraduate course for ophthalmologists. After holding postgraduate courses for seven years which have been fairly well attended by physicians from São Paulo, the interior, and even from neighboring countries, the Ophthalmological Clinic of the Escola Paulista de Medicina instituted the first permanent postgraduate course for ophthalmologists in June of the year 1943. The course will be so arranged that students can begin practical ophthalmology after concluding their studies. The lectures will be given in the presence of patients in the large specialized centers, so that the student can take an active and immediate part in propedeutics and surgery, accompanied by theoretical explanations on diagnostic fundamentals and prognosis. Those who wish to take only part of the course may do so after arrangements have been made with the secretary. The course can be started by the student at any time, thus making it easier for visitors from the interior to make a profitable stay in São Paulo.

Mexico City. The Hospital Infantil in Mexico City was opened to the public on May 29, 1943. This building is the second to be completed in the new Medical Center now under construction. The building is of concrete and has six floors, with a capacity of 550 beds for children of all ages, from newborn infants to children 14 years of age. There is an ophthalmologic department as well as departments of pediatrics, orthopedic surgery, and so forth. The unit will be used also as a teaching department for the medical school of the National University, and has lecture rooms, classrooms, and students' laboratories located in the building. The Hospital is under the direction of Dr. Federico Gomez, a prominent pediatrician, who has been interested for many years in the construction of a children's hospital in Mexico City.

In the September-October number of the Transactions of the American Academy of Ophthalmology and Otolaryngology, Dr. Harry S. Gradle has an article on the history of the Pan-American Congress of Ophthalmology since its inception in 1939 to the first meeting of the Congress in Cleveland, Ohio, on October 11 and 12, 1940. The meeting followed directly upon the close of the meetings of the Academy. The article ends with the enumeration of the 12 fellowships granted by the Kellogg Foundation, which in normal times will be allotted to the different countries as follows: Argentina, 4; Bolivia, 1; Brazil, 6; Chile, 2; Colombia, 1; Costa Rica, 2; Cuba, 2; Dominican Republic, 1; Ecuador, 1; El Salvador, 1; Guatemala, 2; Haiti, 1; Honduras, 2; Mexico, 2; Nicaragua, 1; Panama, 2; Paraguay, 1; Peru, 1; Puerto Rico, 1; Uruguay, 1; Venezuela, 1.

Guatemala. The Girl Guides help in the local sight-saving activities by teaching the Braille method and conducting a clinic for the blind. A free clinic, in charge of Dr. Pacheco Luna, is shortly to be opened in the Casa del Niño. The 800 children who attend that institution will have their eyes examined when they enter at the age of one year, and regularly thereafter until they leave at the age of seven years. Both of these institutions are supported by the International Association of Lions.

Societies

Second Pan-American Congress of Ophthal-

mology, scheduled to be held in Montevideo, Uruguay, November 4 to 9, 1943, has been postponed on account of conditions entailed by the war until the year 1944.

Brazil. Two ophthalmological societies have renewed their Board of Directors for 1943-1944. The Ophthalmological and Otolaryngological Society of Rio Grande do Sul elected Dr. J. G. Valentím, president; Dr. G. Torres, vice-president; Dr. Alfredo Schermann, first secretary; and Dr. J. P. Correa Soares, treasurer.

The Brazilian Society of Ophthalmology, Rio de Janeiro. At the December, 1942, meeting of the Society, the following papers were presented: Myopia due to the use of sulfanilamide, by Prof. Paulo Pimentel; A case of retinal changes in tuberous sclerosis, by Dr. Edilberto Campos.

The São Paulo Ophthalmological Society. The following papers were presented at the February, 1943, meeting: Therapeutic suggestions in three cases of uveitis, by Dr. Candido da Silva; (1) Anti-infectious action of the sulfanilamide drugs on the eyeball, and (2) Epidemic kerato-conjunctivitis and its actual importance, by Dr. Moacyr E. Alvaro.

Mexican Society of Ophthalmology and Otolaryngology. At the September, 1943, meeting of this Society the following papers were presented: The problem of pupillary occlusion and its treatment, by Dr. H. Fernandez Isassi; The treatment of cancer of the larynx, by Dr. L. Vaquero.

Personals

A number of students from Latin America are now studying ophthalmology in the United States for periods of six months to one year. Those known to the editor are: In New York—Drs. Ed. Garduña of Mexico, now at the New York Eye and Ear Infirmary; Drs. Scapini of Costa Rica, Vasquez of Paraguay, and Olmos of Mexico, at the Eye Institute of Columbia University; and Dr. Silva, in Chicago, at Dr. Gifford's clinic.

Dr. Conrad Berens, secretary of the International Congress of Ophthalmology, has been appointed to two new posts: Professor of clinical ophthalmology at Columbia University's College of Physicians and Surgeons, and Civilian consultant in ophthalmology to the Office of the Air Surgeons.

NEWS ITEMS

Edited by Dr. Donald J. Lyle
904 Carew Tower, Cincinnati 2

News items should reach the editor by the twelfth of the month

Deaths

Dr. Philip A. Delavan, Saint Paul, Minnesota, died September 12, 1943, aged 44 years.

Dr. Homer F. Wonders, Philadelphia, Pennsylvania, died August 27, 1943, aged 66 years.

Dr. Charles A. Wynn, Greensburg, Pennsylvania, died August 20, 1943, aged 74 years.

Dr. William B. Anderson, Brownwood, Texas, died July 6, 1943, aged 80 years.

Dr. William K. Foote, Omaha, Nebraska, died July 22, 1943, aged 72 years.

Dr. John J. Finerty, Derby, New York, died September 18, 1943, aged 77 years.

Dr. Charles H. McArthur, Rome, Georgia, died August 2, 1943, aged 47 years.

Dr. Walter S. Moyer, Sayre, Pennsylvania, died July 18, 1943, aged 70 years.

Dr. George H. Bell, New York, New York, died October 5, 1943, aged 77 years.

Dr. Francis G. Speidel, Washington, D.C., died August 30, 1943, aged 51 years.

Dr. Alexander W. Stirling, Baldwin, Georgia, died August 16, 1943, aged 85 years.

Dr. Walter W. Watson, Philadelphia, Pennsylvania, died August 13, 1943, aged 69 years.

Dr. Wilston Johnston, Portland, Oregon, died August 15, 1943, aged 52 years.

Dr. Omar L. Cox, Iola, Kansas, died August 22, 1943, aged 76 years.

Dr. Melvin K. Henry, Philadelphia, Pennsylvania, died August 5, 1943, aged 72 years.

Dr. Frederick H. Martin, Libertyville, Illinois, died August 18, 1943, aged 71 years.

Dr. Edwin S. Moss, Williamsburg, Kentucky, died August 23, 1943, aged 83 years.

Dr. George H. Walker, Winona, Minnesota, died July 2, 1943, aged 63 years.

Dr. John J. Brennan, Worcester, Massachusetts, died August 26, 1943, aged 79 years.

Dr. A. W. Martin, Bogalusa, Louisiana, died October 6, 1943, aged 61 years.

Dr. Frederick W. Mitchell, Houlton, Maine, died September 5, 1943, aged 69 years.

Dr. Simeon A. Daudelin, Worcester, Massachusetts, died August 28, 1943, aged 73 years.

Dr. Carl Barck, Saint Louis, Missouri, died October 2, 1943, aged 86 years.

Dr. Benjamin J. Butler, East Providence, Rhode Island, died September 18, 1943, aged 69 years.

Dr. George R. Gowen, Walla Walla, Washington, died September 2, 1943, aged 61 years.

Dr. Ellwood Harlow, New York, New York, died September 22, 1943, aged 70 years.

Dr. Frank J. Walz, Wilkinsburg, Pennsylvania, died August 2, 1943, aged 72 years.

Dr. Thomas A. Poole, Washington, D.C., died October 5, 1943, aged 72 years.

Dr. Ernst G. Sass, Lidgerwood, North Dakota, died September 15, 1943, aged 73 years.

Dr. Otis F. Simonds, Cleveland, Ohio, died September 26, 1943, aged 61 years.

Dr. David F. Waide, New Orleans, Louisiana, died September 14, 1943, aged 60 years.

Dr. Edwin W. Grubb, Akron, Ohio, died September 23, 1943, aged 73 years.

Dr. Albert J. Guerinot, Pittsburgh, Pennsylvania, died September 18, 1943, aged 66 years.

Dr. Edwin S. Leach, Junction City, Kansas, died September 15, 1943, aged 76 years.

Dr. John M. McGrath, Saint Louis, Missouri, died September 21, 1943, aged 48 years.

Dr. Joseph E. Miller, Los Angeles, California, died September 13, 1943, aged 54 years.

Dr. Samuel J. Ottinger, Philadelphia, Pennsylvania, died September 15, 1943, aged 80 years.

Dr. James L. Rogers, Spokane, Washington, died September 3, 1943, aged 65 years.

Miscellaneous

The Rochester Orthoptic Center (approved by the Monroe County Medical Society) offers a course for orthoptic technicians. Young women desirous of making orthoptics a career and of preparing for the examinations of the American Orthoptic Council may get full particulars from Mrs. Margaret Lundean, Orthoptic Technician, 31 North Goodman Street, Rochester, New York.

The McMillan Hospital was opened for eye, ear, nose, and throat patients on October 15th. This is the only specialty hospital for diseases of these organs in Saint Louis. The present capacity is 105 beds. Two additional floors are to be used at present for neuro-psychiatric patients. These contain 55 beds. This space may be available in the future for ophthalmic and otolaryngologic patients if it is needed.

The first floor and the basement of the building have been occupied by the ophthalmology and otolaryngology departments, respectively, for 13 years. The second floor contains four operating rooms and one large lecture room. The 3d and 4th floors are now used for neuropsychiatry and the 5th, 6th, and 7th floors for eye, ear, nose, and throat. The six floors above these are for research laboratories of the Oscar Johnson Institute, internes' quarters, occupational therapy, squash courts, and offices of departmental chiefs.

The fifth floor is the ward floor of 49 beds. There are one 10-bed division, two 7-bed, two 6-bed, one 5-bed, and four 2-bed rooms for semi-private patients. The sixth floor is for

The McMillan Hospital and the Oscar Johnson Institute.

semi-private patients and the seventh for full private patients.

The Gill Memorial Eye, Ear, and Throat Hospital will hold its eighteenth annual spring graduate course in ophthalmology and otolaryngology beginning April 3, 1944. The following will participate in the program: Drs. Paul Holinger, A. C. Hilding, M. H. Lurie, Beverly Douglas, A. D. Ruedemann, Harold Falls, Raymond Ingalls, Milton Berliner, David G. Cogan, Paul Chandler, Eugene Blake, J. S. Shipman, Major Jerome Hauser, Admiral Ross T. McIntire, and Surgeon-General Thomas Parran.

The next examination held by the American

Board of Ophthalmology will be April 1, 1944, in Chicago.

The Department of Ophthalmology, George Washington University School of Medicine, held its semiannual meeting on December 4th presenting the following program: "Anomalies of the discs" by Drs. William T. Davis and Ernest A. W. Sheppard; "Pemphigus conjunctivae" by Dr. Edgar L. Goodman; "Bilateral amblyopia following crushed chest" by Dr. Ronold A. Cox; "Retinal detachment in childhood" by Dr. Frank D. Costenbader; "Foreign body in cataractous lens located by Vogt X-ray technique" by Dr. Richard W. Wilkinson; "Melanoma of the choroid" by Dr. Sterling Bockoven; and "Hypertensive retinopathy" by Dr. Carmon R. Naples. Colonel Frederic H. Thorne (MC), U.S.A., spoke on "Military aspects of ophthalmology," and Dr. Davis, professor of ophthalmology at the medical school, gave an illustrated lecture on "Differential diagnosis of the vertical motor anomalies."

Societies

At the eighth annual meeting of the Industrial Hygiene Foundation held at the Mellon Institute, November 10th and 11th, Dr. Charles F. Kutscher, Pittsburgh, spoke on "How the inhalation of some chemicals affects the eye."

The first of a series of postgraduate conferences, held by the eye section of the Philadelphia County Medical Society on November 4th, consisted of a discussion of "The diagnosis of corneal diseases" by Dr. Alfred Cowan and a paper on "Practical points in the refraction of the eye" by Dr. Sidney L. Olsho. The following ophthalmologists will participate in the series: Wilfred E. Fry, Isaac S. Tassman, George F. J. Kelly, Edmund B. Spaeth, Walter I. Lillie, James S. Shipman, Francis H. Adler, and Louis Lehrfeld.

The twenty-sixth meeting the Reading Eye, Ear, Nose, and Throat Society was held Wednesday, November 17, 1943. Dr. Warren S. Reese of Philadelphia read a paper on "Remarks concerning ophthalmic surgery." It was announced that Dr. Pearl E. Hackman had been commissioned Surgeon Reserve in the U. S. Public Health Service. Her rank is equivalent to that of a Lieutenant Commander in the Navy.

The regular meeting of the Washington, D.C., Ophthalmological Society was held on November 15, 1943. The guest speaker was Dr. James S. Shipman of Wills Eye Hospital, Philadelphia, who discussed "Treatment of retinal detachment." Case reports on "An unusual case of corneal dystrophy" and "Subconjunctival dislocation of the lens" were presented by Dr. Joseph Dessoff and Dr. J. B. Peebles, respectively.

The new officers of the Society are Dr. Sterling Bockoven, president; Dr. Harold R. Downey, vice-president; and Dr. John R. Lloyd, secretary-treasurer.

The Ophthalmological Society of Egypt, to encourage scientific ophthalmic work, has offered the grant of a prize, the ophthalmic gold medal, for the most valuable contribution brought up before the Annual Congress of the Society, according to a recent bulletin from the headquarters in Cairo.

Personals

Dr. Daniel B. Kirby, professor of ophthalmology, New York University, was guest speaker at the October meeting of the Cleveland Ophthalmological Club. His subject was "Cataract surgery" and the lecture was illustrated with beautiful lantern slides and movies.

Dr. Michel Loutfallah announces the opening of offices for the practice of ophthalmology at 1826 State Street, Santa Barbara, California.

Drs. H. Rommel Hildreth and Carl C. Beisbarth were recently promoted to assistant professors of ophthalmology at the Washington University School of Medicine, Saint Louis.

Dr. Peter C. Kronfeld was appointed associate professor of ophthalmology at the University of Illinois College of Medicine, and also director of education (eye) in the Illinois Eye and Ear Infirmary.

Western Reserve University School of Medicine, Cleveland, celebrated its one hundredth anniversary the week of October 24th. Among the guest speakers was Dr. Daniel B. Kirby who presented a paper on "One hundred years of progress in cataract surgery."